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ON RETINITIS PIGMENTOSA AND ALLIED DISEASES.

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(*Continued from p.* 56.)

On Some of the Exciting Causes of Retinitis Pigmentosa.

Retinitis pigmentosa is the result of a tissue liability present, though seldom manifest, at birth, and known to be so often hereditary that we may be sure it is so also in many cases where the proof is wanting. It is probable that the first changes will be found in the coats of the small choroidal arteries, but this can be neither proved nor disproved until an eye in the early stage of the disease has been fully examined. In the specimen examined by Perrin and Poncet, there were acute inflammatory changes in the choroid, spoken of as metastatic, attributable to the malignant scarlet fever from which the patient (a man aged 21) died, a few days after his eyes had been ophthalmoscopically examined; and these changes masked any choroidal vascular changes that might have existed in relation to the man's retinitis pigmentosa. On microscopical examination pigment granules were found in and amongst the cells of the outer granular layer and the most external part of the outer molecular layer of the retina, but none had reached to the inner retinal layers and consequently none of the retinal vessels were pigmented; Müller's fibres showed some thickening. The man had been night-blind "from birth;" when seen (aged 21) his V. was 12/40 with R., 5/40 with L., Fs. much contracted, colour vision normal, the retina around the disc opalescent and, with the exception of a very few, isolated, stellate dots (microscopical sections of which were not obtained), no pigment whatever was visible under

searching and repeated ophthalmoscopic examination; his father and some other male relations had suffered from night-blindness. The clinical diagnosis of "Retinitis pigmentosa *sine pigmento*" was given.*

We know from the experiments of Wagenmann and others that section of one or more of the posterior ciliary arteries is followed by opacity passing into atrophy and finally pigmentation of the overlying portion of retina; the pigment being derived from the pigmented epithelium and the whole change being due to the atrophy of choroid following permanent interception of its blood supply. Studer† examined the eye of a woman, aged 26, who had undergone optico-ciliary neurectomy a year before enucleation for pain following operation for uveitic cataract, and found the cells of the pigmented epithelium in some parts of the globe not only disturbed but much increased both in number and size, portions of the retina pigmented and the arteries of these portions thickened, and the chorio-capillaris of the corresponding regions either hyperœmic or atrophied. The eye was examined ophthalmoscopically three weeks before the neurectomy and no pigment changes found. If choroidal collateral circulation be quickly established the overlying retina may recover.‡ Studer quotes from Schlodtmann several cases in which the ophthalmoscopic appearances are described at periods from three weeks to seven months after operations for removal of tumours behind the eyeball; pigmentation of the retina was a marked feature in all.

Pigmentation anatomically resembling that of retinitis pigmentosa is often found in eyes that have been blind for a long time from disease of the anterior segment of the globe;

* Poncet, An. d'Oculistique, vol. lxxiv, p. 234 (1875), with illustrations; and Atlas des Mal. profondes de l'Oeil (jointly with Perrin), Pl. XLIX (1879).

† Studer (Theodor F.), On Pigmentation of the Retina after Optico-ciliary Neurectomy in Man, Archives of Ophthalmology, vol. xxxv, pp. 333—348 (1906), from the German Edition (with plates), vol. liii (1905).

‡ Knapp diagnosed Embolism of posterior Ciliary Arteries in two cases, and attributed their recovery without residual changes to the free collateral circulation. A. f. O. xiv, I, 207 (1868).

such eyes are generally glaucomatous (Parsons),* and the retinal pigmentation is a consequence of atrophy of the choroid from the prolonged increase of tension. The condition just described is to be distinguished from another series of cases where chronic glaucoma supervenes in an advanced stage of primary (ordinary) retinitis pigmentosa.†

The practical questions are, *first*; whether an hereditary tendency to disease of small arteries, say those of the choroid, may in favourable circumstances remain latent throughout a lifetime? and *secondly* whether any constitutional conditions can act as exciting causes and evoke the changes leading to retinitis pigmentosa in persons some of whom might otherwise have escaped? An hereditary tendency that remained latent would, or might, account for the atavism not infrequently observed in family histories of retinitis pigmentosa (Figs. 17 to 21, p. 19, and other Figs.). In reference to the second question, such a latent tendency brought into action by hæmorrhage or infectious illness may explain the puzzling cases now and then met with where only one member of a large childship suffers. In fact, the belief is often expressed by sufferers from retinitis pigmentosa that an attack of acute illness, especially an exanthem, has either brought out the family liability or accelerated the disease if it had already begun; illustrative examples will be given further on. There is indeed reason for believing that mere loss of blood may in rare instances rival actual division of ciliary arteries in its results and lead to, or greatly intensify, atrophy of the chorio-capillaris and pigmentation of the retina. Such general causes as toxic illness and loss of blood may be supposed capable of operating either by lowering the power of resistance to an already existing dormant tendency, or by inducing independent vascular

* Parsons, Pathology of the Eye, vol. ii, p. 607 (1905).

† Weiss (E.), Retinitis pigmentosa u. Glaukom., in Vossius's Sammlung zwangloser Abhandlungen, &c., der Augenheilkunde, vol. v, pt. 5 (1903). Schmidhäuser, Retinitis pigmentosa u. Glaucom. Inaug. Dissert. Tübingen (1904). (Professor Schleich.)

disease, and probably both processes may sometimes be at work together. It may even be that certain toxic substances produced in the body have a selective affinity for the choroidal arteries, as quinine has for those of the retina.

The effect of copious rapid hæmorrhage in inducing, or stimulating the progress of, retinitis pigmentosa is illustrated by the following cases:—

CASE 77*.—*Severe uterine hæmorrhage at the climacteric period, causing ocular changes that culminated in retinitis pigmentosa. Similar hæmorrhage, causing acute optic neuritis and temporary blindness in a daughter.*

A lady (P. 27.71), whose health and sight had always been good, was confined to bed for several weeks at the commencement of the menopause, at the age of 49, by severe uterine hæmorrhage. The only local disease was a small polypus at the os uteri. This was about May, 1891. During recovery, but whilst still in bed, both eyes failed; all objects, near and far, were indistinct, but she was not blind; it should be mentioned that she was very slightly myopic. A few weeks afterwards, and again six months later (December) she saw Mr. Higgens, who found V. 6/9 in each eye, with the necessary slight correction, no definite ophthalmoscopic changes, but some loss of the upper part of each visual field. In the spring of 1892, a year after the bleeding, and when greatly distressed and shocked by the death of a son, one eye, the L., failed again rather suddenly, and at the end of June its V. was only 6/36. About six months later (October, 1892), when I first saw her, V. of this (L.) eye had improved to 6/12, but objects were distorted, and in addition to a few roundish opacities in the vitreous there was extensive, shallow, transparent detachment of the lower part of the retina reaching just up to the yellow spot, greatest depth, 8 D.; and patches of pigment, accompanied in some places by whitish deposit, were seen in or beneath the detached portion. R. 6/9 with − 1 D., and fundus noted as normal. I did not measure the fields. No complaint of night-blindness.

* Numbering continued from Figure or Case 76 at pp. 37, 38 of the present volume.

Soon after the above date (October, 1892) the L. became slowly worse, and the R. began to fail. I did not see her again until 1897 (aged about 56); the L. had then been quite blind for some time and the R. though still able to see 6/12, had extreme concentric contraction of field (10° to 20° only), and was very blind at night. The ophthalmoscopic appearances were now almost alike in both eyes, and had entirely altered since 1892; extensive pigmentation of retinæ, denudation of pigment epithelium, sclerosis of choroidal arteries—the trunk vessels showing thick white coats—shrinking of retinal arteries, discs pale and waxy-looking, a few opacities in each vitreous, and in R. a small posterior polar opacity of lens; in L. no trace of retinal detachment, but the whitish masses still present between retina and choroid. In 1902, Mr. Higgins found V. of R. still 6/12, and field not smaller, and in 1904 the report was "about the same."

One of this lady's daughters (P. 42.66) suffered from partial loss of sight in both eyes at the age of 23, after copious bleeding from a uterine fibroid; she recovered V. of 6/5 (em. refraction) in each eye, but when I saw her three months later, there was some loss of F. down-out in the L. eye; and both discs were pale, hazy, and a little swollen, and in the L. the arteries somewhat shrunken.

In the case of the mother just described, the choroidal sclerosis and retinal pigmentation were not found until several years after the uterine hæmorrhage. But the temporary failure of both eyes soon after the bleeding, followed within a year by detachment of retina in one eye, supplies a link, and the phenomena as a whole may fairly be ascribed to disease of choroidal vessels, either initiated or much increased by the loss of blood. That something analogous happened to the daughter is extremely interesting as evidence of the inheritance of very localised peculiarities of structure.

CASE 78.—*Rapid permanent failure of sight with night-blindness after severe hæmatemesis. Advanced retinitis pigmentosa found when first examined several years later.*

George Edmunds (T., O.P., iv, 52), aged 54, a powerfully built man, formerly an engine driver on the South-Western

Railway, came to St. Thomas's Hospital in May, 1882. He was then 54, and said that his sight had been quite good until the events narrated below; but we may take it as certain that the retinitis pigmentosa had been coming on quietly for years before. Married 27 years; no reason to suppose he had had syphilis.

Seven years ago, when about 47, was seized with vomiting of blood whilst mending his engine pumps. The attack weakened him, and when he returned to work he gave up night-duty and drove his engine only by day. Six months afterwards, and again several months later still, the bleeding recurred more or less, and after he had been in bed for a week or two on this account, "something burst again" one morning, and "the blood came up like a pump, two bowls full." During that day his sight failed, and for a few hours he seemed to be in a white fog, and could see no objects; the sight then improved, and kept about the same till we saw him some seven years later, but he never drove his engine again. When seen, May 22, 1882, aged 54, V. of R. was 6/18, L. 6/36; not improved by glasses; pupils acted badly to light; fields much contracted; a small opacity at posterior pole of each lens, and a few round opacities in each vitreous; extreme pigmentation of retina most intense at periphery, but extending in slighter degree almost to the Y.S.; the pigment was in variously shaped spots, round, annular, angular, linear; not a trace of choroidal atrophy, but disturbance of epithelium visible in some places; O.Ds. palish and hazy; retinal arteries moderately shrunken; colour perception slow, but no confusions; hearing good.

In a parallel case by Hutchinson,* a gentleman whose sight had been failing slowly since early life from retinitis pigmentosa underwent, when about 50, and with a year's interval, two severe amputations for diseased bone, the first through the thigh, the second at the hip joint, and on the second occasion he nearly died from secondary hæmorrhage. After each operation he had been "in a mist" for a time, and after the second this was worse, "and he never regained his former sight." Several of his male relatives had had retinitis pigmentosa.

* Hutchinson, Archives of Surgery, vol. ix, p. 182 (Case 32), 1898; and *ibid.*, vol. i, p. 92 (Case 11), 1890.

Mr. Hutchinson adds "it is quite probable that severe losses of blood might be felt by structures already degenerate and with contracted arteries, and not unlikely that such influence might be in some degree permanent."

The patient whose fundus is depicted by Jaeger* in his Hand Atlas was an elderly plethoric man, who assigned the failure of his sight to a period of several years, during which he was losing blood regularly from hæmorrhoids. The appearances, as described in the text, were characteristic of ordinary retinitis pigmentosa, but the illustration is rather atypical, and the history of failure of one eye before the other (if it can be accepted) is unusual.

CASE 79.—*Very severe retinitis pigmentosa, leading to total blindness within about* 10 *years of recognised onset. Uterine hæmorrhage assigned as the cause. Syphilitic arteritis probably contributing.*

Eliza Leeks, an out-patient at St. Thomas's Hospital under Dr. Payne's care, and transferred by him to me (October, 1880), aged 47, married, and had had 15 children, and when 39, about eight years ago, a miscarriage. At the miscarriage she lost a great deal of blood, amd both she and the daughter who brought her to the hospital dated the failure of her sight from this flooding; both eyes failed equally. After a time she began to complain of seeing worse in the twilight, and the sight had become much worse during the few months before she came for advice. In October, 1880, aged 47, the could barely see her way about; media clear, the discs yellowish and hazy, retinal arteries contracted to the most extreme degree; retina extremely pigmented from periphery almost to disc, but the Y.S. free; pigment more blotchy than usual, perhaps, only because there was so much of it; no choroiditis anywhere. When seen again three years later she had no p.l. When three years old she had scarlet fever, and had been very deaf ever since; and she had been subject to epileptic fits with tongue-biting since she was about 15. She was one of a childship of nine—five boys, all of whom died before they grew up, three girls (besides herself), all

* Jaeger, Atlas d'Ophthalmoscopie, p. 129, and Pl. XVII, Fig. 79 (1870).

living when the notes were made. She was the only one known to have had bad sight or nervous symptoms. Parents were not consanguineous. She herself had had 15 children, besides at least one miscarriage. One died of consumption, five others died young, and nine were living and reported healthy; one of these, a daughter, was examined, and her eyes found normal. Mrs. L. was in a condition of active tertiary syphilis when we saw her, with nodes on the head and clavicle, and a perforation of the palate. But the eye changes were not like those of syphilis, and we may conjecture that retinitis pigmentosa, beginning early in life, was made rapidly worse by acute, passing into chronic, anæmia of the choroid, due to the combined effects of local syphilitic arteritis and severe systemic hæmorrhage.

Retinitis pigmentosa apparently caused or accelerated by acute exanthemata or other illnesses.—A number of such cases are on record, and no doubt many others have been seen. The most convincing example that I have met with is Case 80 below, but I shall give several others, and refer to some of the best of those that have been published. In many of the hitherto described cases of this class there are central changes either in addition to, or sometimes to the exclusion of, the typical equatorial pigmentation, and in such we find damage to central vision as an early symptom. Cases of retinitis pigmentosa, with relatively severe central loss, have long been known. De Wecker, in 1868, stated that in such cases the retinal disease progressed more quickly than usual, and that they were specially liable to mental complications; I am not aware that this view has been either confirmed or refuted. The alleged influence of acute exanthems on the course of retinitis pigmentosa was briefly mentioned by Leber in the 1st Edition of Graefe-Saemisch (Vol. V), but was not connected with any particular localisation of the changes.

If an acute infectious disease can bring about changes in the choroidal vessels, the same may be true of such chronic fevers as syphilis and tuberculosis. The occasional difficulty of making the differential diagnosis between ordinary or "true" retinitis pigmentosa and chronic progressive diffuse

choroido-retinitis in the subjects of syphilis is well known; and possibly a few of these cases might more correctly be designated retinitis pigmentosa, brought out by syphilitic narrowing of choroidal arterioles. A corresponding position might be claimed for tubercle, but no sufficiently precise data exist as yet for testing the question. There is no doubt, as Dr. R. J. Smyth, of Guildford, pointed out to me in connection with one of his pedigrees of retinitis pigmentosa, that a history of phthisis is common in such families, but the time is not ripe for any attempt to treat the question statistically.

CASE 80.—*In a family of* 10 *children*, 5 *have scarlet fever in early life, followed by retinitis pigmentosa*, 2 *die of the fever*; 3 *escape the fever and have no eye disease.*

In the H. family (P. 6, 89, etc.) we can scarcely doubt that scarlet fever in early childhood was the exciting cause of progressive retinitis pigmentosa in children with unstable nervous system and with consanguinity on the maternal side. In most of the victims the retinal changes differed somewhat from the common form, but in one they were perfectly typical, and, in general, the progressive character of the disease was well shown. The family was under my notice between 1882 and 1904. In the following account the 10 siblings are placed in their order of birth :—

1. ♀ had scarlet fever at 3 years of age ; her sight failed in childhood, after the fever, but the exact time of onset uncertain; got slowly worse for a time, but exact state of vision when she died of bronchitis, at 50 (1899), is not known. I saw her in 1882, aged 33, with V. less than 6/60, and with + glasses 6 J. held close ; Ps., acted a little ; Fs. of full extent ; colour vision normal ; sight best in bright light ; epithelial disturbance all over central area in both eyes, worse in R. ; retina at periphery rather hazy but showing no pigmentation ; O.Ds. rather pale and hazy ; arteries too small. A sleep-walker, eccentric, suffered at times from delusions, and is said to have had Graves' disease at one time. Teeth not suggestive of hereditary syphilis.

2. ♂ born a year after 1. Had scarlet fever when he was 8 years old, *and on the same occasion the five younger children, being*

all that were then born, caught the disease. The sight of No. 2 began to fail just after the illness and got gradually worse. At 22, was able to take his degree at Cambridge, but when I examined him in 1904, aged 54, he could only spell out very large letters with a hand-lens, and for many years had been unable to read ordinary print. He disliked bright light, could go about alone quite easily, and was not night-blind; colour-vision good. Changes in general like those in No. 1, but there were some small collections of pigment on the diseased central area, and sclerosis of some choroidal (short ciliary) arteries. Mental condition good.

3. ♀ two years younger than 2; failure of sight is dated from the scarlet fever. At 23, V. still good enough to be benefitted by glasses for reading; at 26 she married. At 30 (1882) I saw her with Mr. Stanford Morton; V. eccentric, about 16 J.; colour-sense quite good; dislikes bright light; large surface of epithelial loss, with slight pigment collections, at each Y.S. area; border well defined and sinuous; arteries doubtfully shrunken; O.Ds. pale and hazy; refraction H. As. In 1904, reported to be "about the same" and to have three grown-up children with perfect sight.

4. ♀ a year younger than 3; sight became bad, as in the others, very soon after the fever. At 28, could still see well enough to be a governess, and was not blind when she died, at 43 (1897), of heart disease and dropsy; but latterly her mind had failed. I am not sure whether her eyes were ever examined.

5, ♀, and 6, ♀, were aged 4 and 3 respectively when they had the scarlet fever; both died—5 from the fever and 6 from consecutive diphtheria.

7. ♀ was 10 months old at the time of the family epidemic; she was ill at the same time, "either of scarlet fever or measles," and afterwards had "abscess in the ear." Precise date of failure of sight not known; as a child, "could see to read fairly well"; began + glasses at 14. When seen at 24 (1882), well-marked nystagmus, sight worse in bright light, loss of nasal third of each field, with her + sees letters of 6 J. eccentrically. Very typical advanced retinitis pigmentosa extending back to O.D.; O.Ds. more hazy and arteries much smaller than in No. 1, examined at same date. Emotional. In 1904 (aged 36), reported to be nearly blind.

Note that she had the fever at a much earlier age than any of the others, and that her eyes were the worst of any. The nystagmus points to sight having been bad in infancy, probably very soon after the illness.

8, ♂, 9, ♀, and 10, ♂, were born after the scarlet fever outbreak, and never had the disease. In 1904, they were aged from 44 downwards, and all enjoying perfect vision. I had examined the eyes of No. 9 in 1886, when she was 24, and found them healthy; she is extremely hysterical and has more than once been out of her mind.

It may be noted that the mental powers and balance of the three living males (2, 8, and 10) are very good, perhaps rather above the average. The father took to drinking late in life. His wife and he were not consanguineous; she, a healthy woman, was the offspring of "distant" cousins.

CASE 81.—*Atypical retinitis pigmentosa, ascribed to prolonged fever (probably typhoid) Case watched for* 12 *years. No other cases in the family.*

Frederick Mayhew (M., O.P., i, p. 8), seen at Moorfields in 1882, aged 24, and again in 1892 and 1894. During this period V. sunk in R. (the better eye) from 6/18 and 1 J. with ring scotoma, enclosing clear centre of 10°, to 6/36 and 15 J., with reduction of the clear centre to about 2°. L. was worse than R. in 1882, and did not deteriorate much between then and 1892, when it was about equal to R. The changes described in 1882 had visibly increased by 1894, when the following note was written; the disease is confined to central region and equator, and symmetrical in the two eyes; at equator, chiefly in nasal half, coarse, elongated pigment deposits, many of them conspicuously encrusting the veins; all over the Y.S. area pigment epithelium disturbed, as if the individual cells had joined into small groups, leaving some finer granules between; O.Ds. only slight waxy haze; retinal arteries and veins little, if at all, shrunken. Periphery of fundus free. When 11 years old he had a severe illness, called "slow fever"; his mother says his sight was lost for three weeks during the illness, that it then improved for a time, but soon began to go off again, and has steadily got worse ever since. His own earliest remembrance about it is that he

found he could not see to pick up small things, such as marbles, nor see what he looked at, and that he began to see worse in the twilight. No other cases were known in the family, sight quite good in the mother, aged 60, and her parents, and in the father, who died at 46; nothing known, however, about father's parents. No consanguinity; no family deafness, malformations or defects. Patient shows no signs of hereditary syphilis, and denies having had any venereal disease; he is the third born of 9, 6 ♂, 3 ♀, all living and well, and not one of them, except himself, has ever complained of defective sight.

CASE 82.—*Atypical retinitis pigmentosa. Recent rapid deterioration after pneumonia.*

Charles North (O.P. letters), cook, seen at Moorfields, March, 1888, aged 35. Slight failure of sight two or three years ago, but no definite history of night-blindness; could read well until six months ago, when vision failed after a six weeks' illness called "pneumonia and congestion of the lungs," and in two months was as bad as now. Had "typhoid" 15 years ago; denies venereal disease, and shows no signs of inherited syphilis. Has never heard of bad sight in any relations; parents were not consanguineous. Married, wife healthy, and has had seven children, all ♂; 1st, 3rd, and 6th living, aged 10, 5, and 2; the other four died in infancy. On admission, patient's sight, 6/36, H.m. 1 D.; with +2 D. reads 14 J. slowly. Fs. of almost full size, but with a ring scotoma about 15° wide, with clear centre of about 10°. Changes exactly symmetrical in the two eyes; (1) a complete zone of pigmented retina enclosing central area, and coming nearly up to O.D., but not extending further anteriorly than the equator, with denudation of corresponding pigment epithelium; (2) an irregularly-shaped area of epithelial disturbance at Y.S. without pigment accumulations; this area more or less definitely separated from the pigmented zone; O.Ds. rather hazy, arteries of fair size, many veins encased by pigment.

E. Schmidt, in his Dissertation (1890), mentions male twins (his Cases 16 and 17) who saw well till they were two years old, when they had measles and diphtheria, and, according to the history, became blind within five months. When examined at

the age of nine both boys were blind of both eyes except in a small sector in the temporal field; changes alike in both, haze of O.D., shrinking of vessels, and fine pigmentation of retina.

Gonin (1901) (Case 3), a girl of 14, with abundant typical retinitis pigmentosa, nystagmus, and very bad sight, in whom the symptoms were dated from typhoid fever at the age of two years. She was the first born of seven, and all the other six (some ♂, some ♀) were said to have perfect sight. No consanguinity, and no cases known amongst relations.

Hersing, in 1872, reported the case of a man, aged 26, whose sight had failed somewhat rapidly after typhus fever in which the cerebral symptoms were severe. Marked night-blindness with some defect of F., but good central V.; at the periphery fine choroido-retinal changes with but little pigmentation; O.Ds. and retinal vessels normal.

Derigs (1882) Case 16, ♂; failure of sight dated from scarlet fever at six years old; saw well till then. Seen at 16 with severe night-blindness and much contracted Fs.; typical retinitis pigmentosa at periphery; elsewhere diffuse atrophy of pigment epithelium and choroidal stroma, but Y.S. exempt. At above date all his seven siblings had perfect sight; but in 1890 one of his brothers, 10 years his junior, was seen at the age of 14 by E. Schmidt, with much contracted Fs. and scanty peripheral pigmentation of retina (E. Schmidt's Case 29). No consanguinity.

In a case by Mellinger (1891) typical retinitis pigmentosa of some, probably long, standing in a woman, seems to have been made worse by an attack of influenza at the age of 51, night-blindness, not previously troublesome, becoming very noticeable soon afterwards. Her four siblings and both parents said to see well. No consanguinity.

Wider (1885) gives three cases in which scarlet fever, and three others in which "typhus" (probably typhoid), was believed by the patient to have brought on the eye disease. The particulars, are very briefly given, and the presence of retinitis pigmentosa in the sisters of one patient, of deafness in the siblings of another, and of congenital absence of arms in a near relation of a third reduces their value for the present purpose.

A similar remark applies to Herrlinger's (1899) Case 7 attributed to measles; both this girl's siblings also had retinitis pigmentosa. For the same reason his Cases 57 and 58 are inadmissible.

The above are only a few of the published cases where the changes leading to retinitis pigmentosa are supposed to have been set in action by an attack of infectious illness. The list might easily be lengthened, but the cases quoted are sufficient to draw attention to the importance of more extended and detailed enquiries in future.

Retinitis Pigmentosa Affecting only one Eye.

In the cases that follow there can be no doubt that the disease was anatomically the same as common binocular retinitis pigmentosa. They can be explained at the present moment only by supposing that the vital endowments of the choroid are sometimes different from birth in the two eyes, an assumption by no means improbable, although unproved. It will be noticed that there is no family history of the disease in any of these uniocular cases. In selecting them I have excluded all in which there is any reason for suspecting syphilis either in the history or the ophthalmoscopic appearances. The only example that I have seen in my own practice is the following:—

Case 83.—Miss A., 30, December, 1894 (P. 32, 243). Perfectly typical and very abundant, retinitis pigmentosa affecting R. eye only; the belt of pigmentation is complete and its anterior limit cannot be reached in ophthalmoscopic examination; O.D. and retinal vessels characteristic; V. with – 2 D. 6/18; eye diverges; field not measured. L. V. 6/5, slight H. As.; ophthalmoscopic appearances normal everywhere. She knew that the R. had been "weaker" in sight than the L. even in childhood; no other history. Red hair, fair complexion, teeth very good. Family history was not investigated.

Pedraglia, in 1865, saw a man aged 36, well, strong, and apparently healthy, whose R. eye had been divergent from earliest childhood; the F. was contracted and V. only 6 J. held

close; media clear; fundus much pigmented from periphery to Y.S. and border of O.D.; O.D. yellowish; retinal vessels only half as large as in other eye, and choroidal vessels sclerosed; in 1891, Deutschmann had the opportunity of examining the eye anatomically. The L. eye had normal sight, full field and fundus perfectly healthy. Parents not consanguineous and no other cases known in any branch of the family. Hereditary syphilis not mentioned.

De Wecker's* case (1870). Girl aged 15, whose L. eye had only bare p.l. and was believed by her parents never to have had sight; it diverged—My. $\frac{1}{4}$ (say 9 D.) in each eye, V. of R. = 2/3 with correction; L. not improved. L. fundus advanced typical retinitis pigmentosa, the Y.S. and its immediate neighbourhood being the only part free from pigmentation; pigment epithelium absent over the whole fundus, but no atrophy of choroid; media clear. R. carefully examined after atropine and no trace of pigmentation found anywhere. Syphilis not mentioned. Nothing as to family history or consanguinity.

Baumeister's case is well-recorded and important from the coincidence of deafness of the ear corresponding to the affected eye. (1873.) It was as follows:—

A man of 44 had well-marked retinitis pigmentosa of the L. eye only, with shrunken retinal arteries and atrophic disc; V. only p.l. and the eye had been "blind" since æt. 12. Since childhood he had also been deaf in the L. ear and Dr. Land reported appearances of past inflammation which might have occurred in childhood or even before birth. The R. eye was normal both as to fundus and V. and not night-blind; and there was no deafness of R. ear. His siblings all normal. There seems to have been some doubt whether the parents were related or not.

Derigs's case (Case 27, 1882), not fully reported, was in a woman of 46 who had advanced retinitis pigmentosa of R. only; eye defective from youth and has steadily got worse; V. now 10/100, F. much contracted, posterior polar cataract. A brother has the same disease but whether in one eye or both is not stated. No consanguinity of parents.

* Traité des Maladies du Fond de l'Œil, par L. de Wecker, accompanied by Atlas d'Ophthalmoscopie, par E. de Jaeger, p. 142, footnote. Paris and Vienna, 1870.

Ancke's case (1885) is somewhat atypical. A boy, aged 15, now in good health but has had diphtheria, scarlet fever, measles with dropsy and "typhus"; no signs of syphilis. Parents not consanguineous. R. eye perfectly healthy as to V., F., and fundus. L. said to have been defective for eight years; now V. about 1/3; F. (taken repeatedly during the next two years) considerably contracted by day and much more so in dull light; typical retinitis pigmentosa towards periphery, but equator thickly sown with bright confluent dots; retinal arteries contracted.

Reinecke (1894) gives brief and not quite convincing particulars of the case of a married woman, aged 44. L. eye defective for a long time but no precise history obtained; now V. = fingers, advanced pigmentation. No changes found in R. eye, but V. with + 1·5 D., only 20/100. No syphilis.

Rosenbaum's Case 34 (1900), given very briefly, was that of a patient, aged 20 (sex not recorded), with retinitis pigmentosa and constricted F. in R. only. L. fundus normal. No other particulars.

Case 9 in Gonin's second paper (1902) is one of typical advanced retinitis pigmentosa with very bad V., confined to the L. eye, in a man aged 25; the eye was said to have been bad from childhood and had been examined and pronounced incurable at Strasburg some years before Gonin saw the patient. R. eye normal in every respect except H.As. No consanguinity of parents; no other cases known in relations. Had scarlet fever (? measles) some years ago, but the eye appears to have been bad before that. Four of patient's siblings died young and their father died suddenly at 34; these are the only recorded facts pointing to syphilis. A younger brother of patient had perfect eyes.

I reject Mooren's often quoted case in a woman of 35 (1867) since he expressly states that although visible pigmentation was confined to the L., the R. showed "rather scantily developed" retina and optic nerve and a much constricted F.

Neuffer's case (1893) is also omitted because (1) there was disease of vitreous and perhaps detached retina; (2) a sister had choroiditis; and (3) syphilis is not mentioned.

(*To be concluded.*)

HEREDITARY OPTIC ATROPHY (LEBER'S DISEASE).

By W. Ilbert Hancock.

J. M., male, age 26, consulted me on October 24, 1906, for failing vision in both eyes.

Early in March of that year, when living in Brazil, his sight commenced to fail. He first noticed a mistiness on fixing an object intently, and found that on eccentric fixation the visual acuity greatly improved.

This failure increased very rapidly, and at the end of a fortnight, although he could find his way about without much difficulty, it was quite impossible to read the largest print. No headaches or pain in the eyeballs. On examination:—

R.V. = counts fingers eccentrically, not improved.

L.V. = „ „ „ „

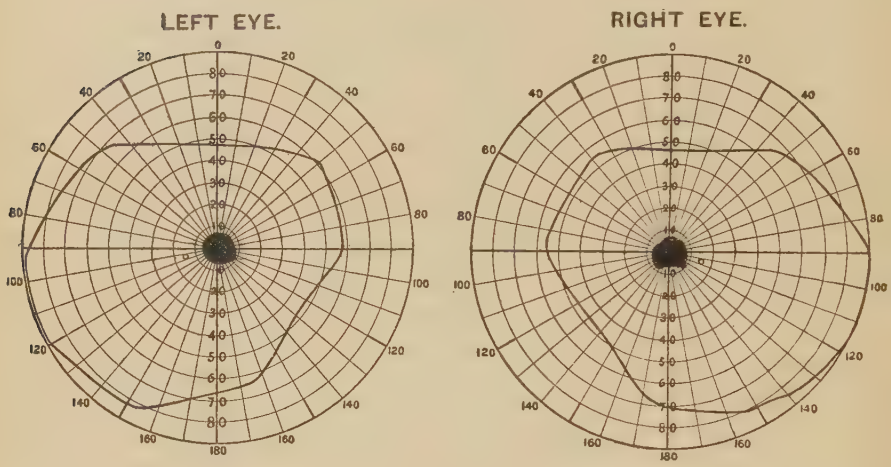
Myopic R. and L. – 2·5 D. sph.

L. pupil slightly larger than R., both reacting sluggishly to light, briskly to convergence. The contraction to light was badly maintained, and after several oscillating movements the pupils became stationary in a semi-dilated position.

On examination of the fundi both optic nerves showed marked pallor, especially in the temporal halves, the nasal halves still retaining a faint pink flush. There was an indefinite haziness of the whole disc and immediately surrounding retina, and the outline was ill-defined, but no patches of exudation or hæmorrhages were present. The physiological cups were filled in. Retinal vessels normal. Maculæ and fundus elsewhere normal. Vitreous clear.

Fields (see chart) show a central scotoma for white within the 10° circle, with normal peripheral limits. Patient is habitually a moderate smoker, 5 to 10 cigarettes a day, but for the last six months he has given it up entirely, having been advised that his trouble was due to tobacco poisoning. He is a total abstainer and leads a very quiet life. No history of syphilis or sexual indulgence. Since 16 years of age he has suffered from occasional attacks of epilepsy, and for the last two years he has been taking

large doses of bromides (90 gr. per diem), which have considerably diminished the frequency and severity of his epileptic seizures.



The patient was very carefully examined by Dr. Fawcett, to whom I am indebted for the following notes on his general condition :—

"J. M., aged 26. Since 16 years of age has suffered from epileptic fits. Parents healthy. No history of any nervous disease in the family.

"At the age of 20 fits became more frequent, occurring at intervals of a month. No evidence of intracranial disease or any organic disease of the nervous system. Heart and lungs normal. Urine normal."

On questioning patient it was found that members of his family for five generations had been attacked by failure of sight, and although the appended family tree (p. 171) with details of the affected members is for obvious reasons incomplete, it shows many points of interest.

Subsequent Progress.—Patient at his first visit was advised to take a prolonged voyage and to refrain from smoking. The bromides were reduced to 50 gr. per diem and Fowler's solution was added.

November 8, 1906. Conditions in *statu quo.*

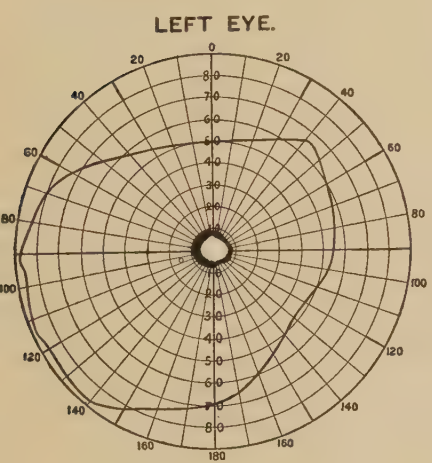
January 30, 1907. R.V.: 4/60 not improved } L.V.: 4/60 „ „ }. A distinct improvement in sight. Haziness of discs and surrounding retina has entirely disappeared. Colour of discs much the same as at first visit. Retinal vessels normal.

June 25, 1907. Great improvement in vision. Patient admits that for the last four months he has been smoking 8 to 10 cigarettes daily.

R.V. $\bar{c}$ – 2·5 D sph. = 6/24. J. 4 picked out.

L.V. $\bar{c}$ – 3 D sph. = 6/24. J. 4 picked out.

The fields show that the scotoma for white has now cleared in the centre, leaving a complete ring (see chart). There is still a relative central scotoma for green and blue, but patient recognises the smallest pieces of red with ease. No enlargement of the blind spot. Discs as at last visit.



September 26, 1907.

R. $\bar{c}$ – 2·5 D sph. = 6/9 full } L. $\bar{c}$ – 3 D sph. = 6/9 partly } J. 1 perfectly, 6/6 partly.

Light sense very defective with Bjerrum's types R. and L. = 3/60 (self = 6/9). The ring scotoma is now incomplete;

a narrow crescentic area above and below in each eye being all that remains. No relative central scotoma.

Patient returned to Brazil in October, 1907.

Description of Affected Members in Family Tree.

CASE I.*—The details of this member of the family who died about 100 years ago at a ripe age are very scanty. It is known, however, that in the third decade of life, his sight failed very rapidly, and that after a prolonged period it greatly improved.

He married and had issue three sons and three daughters. All the sons and one daughter remained single, and were unaffected—two daughters married and had issue (see tree).

CASE II.—Was attacked with marked failure in the vision of both eyes between the ages of 20 and 30. He was a heavy smoker and drinker, and never recovered.

CASE III.—Attacked in exactly the same way as his elder brother in adult life. He was a very abstemious man, moderate in tobacco and alcohol. After from 12 to 18 months he recovered his sight sufficiently to resume his occupation as a business man.

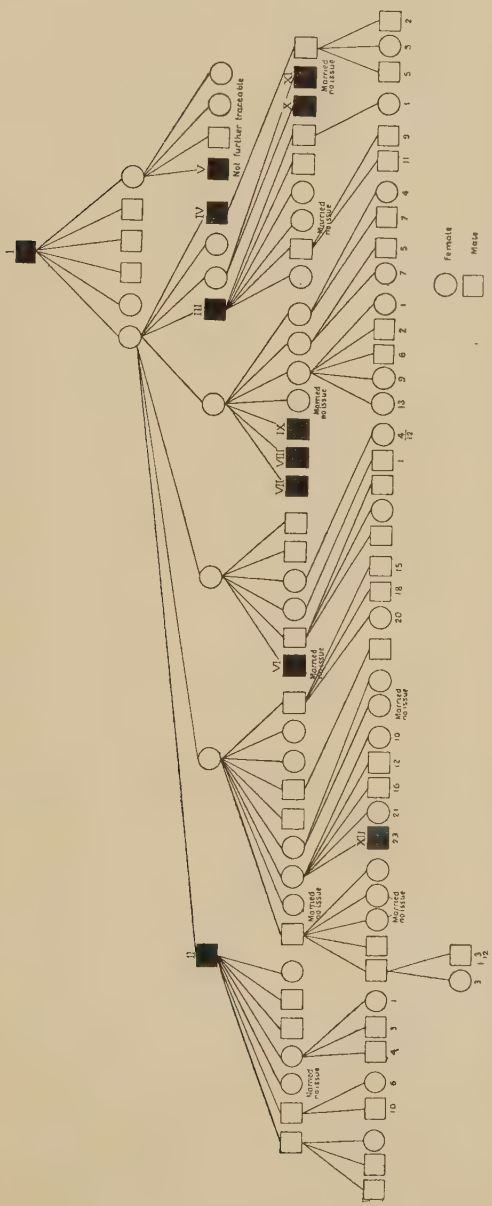
CASE IV.—Attacked when between 20 and 30 years of age in the same way as his two brothers, with the exception that his sight is said to have failed him almost suddenly when working in his office late at night. He was by no means a careful liver, and never recovered his sight.

Although Cases II and IV never recovered, they did not become completely blind; they were incapacitated from doing any work, however, being unable to recognise people, or read print, however large.

The exact age at which Cases I, II, III, IV were attacked is unknown, but there is little doubt that it was during the third decade of life in each instance.

CASE V.—The notes obtainable of this case are very scanty. He was attacked at about the age of 30 and did not recover.

* The numbers correspond to those over the black squares in the diagram.



The black squares indicate affected members. The numbers over them correspond with the case numbers in this paper. The figures beneath the lowest two lines give the present age of these members of the family.

He never lost his sight completely, but was unable to follow any profession. He died in middle life.

CASE VI.—For notes of this case I am indebted to Dr. McNabb of Manchester, who was consulted by the patient.

On returning from a holiday in December, 1906, the vision in both eyes failed rapidly. General health excellent. No specific history. Married, no family. Smokes 2 to 3 ozs. of cigarettes weekly. Takes alcohol in moderation.

March 26, 1907. R. = 5/60, not improved, J. 16. L. = 5/60, not improved, J. 16.

Pupils equal, medium size, reacting to light and accommodation. Temporal half of each disc pale, but fundi otherwise normal. Central colour scotoma for red and green, no scotoma for white. Peripheral field normal. Nervous system normal.

Treatment.—Strychnine, cessation of smoking, minimum of alcohol and prolonged holiday.

August 28, 1907. R. V. = 5/60, not improved, J. 20; L. V. = 5/60, not improved, J. 16.

Large central scotoma for white. Pallor of disc more marked. Veins somewhat tortuous. Arteries small. Peripheral field normal.

Patient is now abroad and in a recent letter his wife states that there is a distinct improvement in vision.

CASE VII.—Acquired the disease at the age of 25. Took no care of himself, being a heavy smoker and drinker. Never recovered, but did not go blind.

CASE VIII.—A fine athlete, attacked at the age of 20 when in hard training for a flat race. Non-smoker and total abstainer. In 12 to 18 months his sight is said to have completely recovered.

CASE IX.—Recorded in detail at the commencement of this paper.

CASE X.—This patient I have had the opportunity of examining. He was attacked by rapid failure of vision in both eyes at the age of 26. The failure commenced as a central

defect, and in two weeks was so bad that he had to be led about, although he was never quite blind.

When attacked he was suffering from bad tertiary syphilis, to which his eye disease was attributed. No history of headaches or pain on movements of the eyeballs. Heavy smoker. Alcohol in moderation. He took a long sea voyage, giving up alcohol, but still smoking to some extent. Eighteen months after the commencement of the attack the sight in each eye commenced to return rapidly, and in two months was as good as it is at the present time.

On November 8, 1906, his age being at that time 41, his condition was as follows:—

R. V. = 6/6, J. 1. L. = 6/6, J. 1. Pupils equal and active. Light sense very defective. With Bjerrum's types R. and L. = 6/60 barely. (Self = 6/6.) R. and L., optic nerves very pale, the temporal halves appearing perfectly white, but the faintest tinge of pink being perceptible in the nasal halves. Vessels, both arteries and veins rather small, but show no sclerosis of their coats.

Slight pigmentary disturbance of choroid to outer side of discs. Physiological cups somewhat filled in. Maculæ and fundi elsewhere normal. Patient complains that although his sight is generally good, he finds difficulty in seeing rapidly-moving objects, *e.g.*, a bird on the wing, so that he has had to give up shooting. He also finds that, if dining off a white plate, he frequently helps himself to salt two or three times. The fields taken with a white object in a good light were quite normal, there being no enlargement of the blind spot, or central, or paracentral scotoma. No relative scotoma. Colour vision normal. Urine normal. Nervous system normal. Patient in robust health.

Case XI.—This patient I have also examined.

His sight failed him in both eyes in April, 1898, when 31 years of age. The onset was rapid, and in 10 days the attack was at its worst. Twitching of the eyelids was the only other symptom of which he complained, and at the time he was working very hard, with late hours and irregular meals, and was much run down. Heavy smoker and moderate drinker. The central vision failed first, and he could always find his way about,

although quite unable to read the largest print. Sight commenced to improve in October, 1898, and continued to do so for six months, at the end of which time it was as good as now. In June 25, 1907, his age being at that time 40, his condition was as follows :—

R.V. = 6/6 most, J. 1. L. = 6/6 most, J. 1.

Pupils equal and active. Light sense very defective. With Bjerrum's types, R. and L. = 6/36 (self = 6/6).

R. and L. discs very white, especially temporal halves.

Outline sharply defined, physiological cups somewhat filled in. Vessels normal. Some pigmentary disturbance of choroid to outer side of disc.

Field taken with a white object in good light perfectly normal. No enlargement of blind spot. Colour vision normal. No specific history. Urine normal. Nervous system normal. Married, no children living. Wife had two miscarriages and suffers from a uterine affection.

CASE XII.—Only scanty notes obtainable. Acquired the disease at age 17. Never recovered, but not blind.

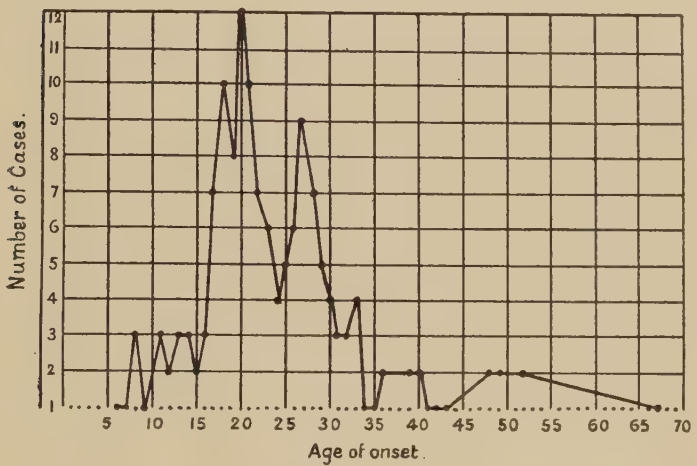
Analysing the above cases, they present several features of interest.

It will be seen that of the 12 members attacked by the disease, six have practically recovered their sight, and one is said to have commenced to improve. This is a most unusual proportion of recoveries, and although such cases are recorded, they are no doubt rare.

Leber, in his Classical Monograph, published in 1871, states that the prognosis is not absolutely hopeless, in so far as the disease is limited to the loss of central vision, but in a later communication considerably modifies this view. Complete blindness is extremely rare, the peripheral field as a rule remaining good. In four recorded cases recovery was complete in one eye only. In the above cases it is interesting to note the prolonged interval of time before the vision even commenced to improve, Cases VIII, IX, X, XI being 12, 9, 18, and 7 months respectively.

With regard to the time of onset, with one exception all the cases acquired the disease in or about the third decade of life, youngest 17, oldest 41.

As will be seen by the appended chart of 150 cases of undoubted Leber's disease taken from literature, this is by far the most common time of life for the disease to commence. Usually, males only are attacked. When, however, females are affected, it would appear, as far as one can judge



from the small number of cases recorded, that in a large proportion of cases they acquire the disease at or about the climacteric. Leber states that the disease is relatively more acute and more severe when affecting females, but this view is questioned by Hormuth in his most admirable monograph, which all interested in the subject should peruse.

True to the type so frequently assumed by other well-known familial diseases, only males are affected in the above family, and in all the cases the disease is transmitted through the female line.

By some authors it has been pointed out that infant mortality is a striking feature in the families affected with this disease.

From the natural disinclination of parents to speak on this subject, it is often difficult to obtain the desired information, but certainly in the later generation of this family, the infantile death-rate has not been unusually high.

In the field of vision depicted on p. 168, the large central scotoma for white, and normal peripheral field, is quite characteristic of the disease.

It was interesting to note that the central portion of the scotoma was the first to clear, and even after the vision had returned to 6/9 there was a distinct ring scotoma corresponding to the extreme periphery of the original central defect. There are several cases recorded in literature in which the central scotoma has been associated with a large defect in the field of vision, and this atypical form has even proved characteristic of several members in the same family.

Consanguinity has played no part, and in no single instance in the above family tree has there been intermarriage with blood relations.

Lastly, it would appear that tobacco and alcohol have little or no influence on the disease. Case VIII was a non-smoker, and said to be an abstainer, and was attacked whilst in strict training for a foot race. Cases IX, X, and XI, all of whom after a prolonged interval recovered, frankly admit smoking and taking alcohol for two or three months before their sight commenced to return, and also during convalescence.

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THE PATHOLOGY OF COLOBOMA AT THE NERVE ENTRANCE.

By George Coats, Curator.

With Plates VI and VII.

Among the rarer congenital abnormalities of the eye, one of the most interesting is that known as "Coloboma of the Optic Nerve" or, better, as "Coloboma at the Nerve Entrance." Ophthalmoscopically, it takes the form of a deep pit, often measuring several times the diameter of the normal disc. Smaller secondary excavations are often present in its floor. The retinal vessels emerge from its depths with a variable arrangement, and bend sharply at its overhanging edge. Frequently, towards the upper half, an ill-defined pinker area contrasts with the pearly whiteness of the coloboma elsewhere, and seems to represent the situation of the papilla.

With regard to the arrangement of vessels, the classification of Caspar* is usually adopted.

(1) The lower part of the coloboma is most deeply excavated, and from it the vessels spring. Their general direction is upwards, so that those destined for the lower half of the fundus must make a sharp bend downwards over the deeply excavated lower edge. The superior vessels, on the other hand, take a more direct course to their destination over the less excavated superior border.

(2) The vessels arise with something like their normal arrangement either from the centre of the coloboma, or from a point towards its upper border.

(3) The vessels emerge separately from one another at some distance from the centre of the excavation, or even

* Inaug. Diss. Bonn, 1887.

quite at its edge. In the latter case the retina appears to be entirely supplied by cilio-retinal branches.

It is customary on clinical grounds to draw a distinction between "Coloboma of the Optic Nerve" and "Coloboma of the Choroid adjacent to the Nerve." In the latter the defect has the more or less triangular shape common in colobomata of the choroid elsewhere, and though it may touch, encroach on, or even engulf the papilla, yet the boundaries of the latter are fairly distinct, and separable from the floor of the colobomatous area. In "Coloboma of the Nerve," on the other hand, an enlargement and excavation of the nerve itself are simulated, although, as stated, some part of the excavated area may be indefinitely distinguishable by its pinker tint or by the distribution of the vessels, as the probable situation of the entering nerve fibres. This appearance may be absent, however, so that the papilla appears to be replaced by a deeply excavated area which may be as much as twenty times the normal diameter of the disc.

The present paper deals especially with the anatomy of "coloboma of the nerve," as above defined, but it will be necessary also to make some reference not only to coloboma of the adjacent choroid, but also to various other congenital abnormalities which would seem *à priori* to be related to these two. Such are, for instance, Fuchs' coloboma or inferior crescent, and holes and excavations within the diameter of the papilla itself.*

* Under the name "peripapillary staphyloma" a condition has been recently described by Kayser, Klin. Monatsbl. f. Augenh., vol. xlvi, p. 76, 1907, in which a more or less normal nerve, with its normal arrangement of vessels, is visible at the bottom of a deep pit in the surrounding tunics. This is not strictly a coloboma, since there seems to be no defect. It is presumably a congenital malformation or ectasia of the ocular tunics, but as I have nothing to add to its pathology, I shall not deal with it here (see Hancock, Trans. Ophth. Soc., vol. xxvii, p. 167, and Verderame, Arch. f. Augenh., vol. lviii, p. 1, 1907.

Historical.

In order that the reader may be in a position to judge for himself on what basis our knowledge of the anatomy of coloboma at the nerve entrance rests, I shall first give a brief abstract of those published cases which seem properly to fall within this category. I shall then report three rare cases which I had the opportunity of examining, and thereafter review the literature as a whole, seek to correlate my cases with those already published, and attempt to deduce some generalisations from the isolated facts. Allowance must be made for the difficulty of giving in succinct form an account of relations which are often exceedingly complicated. In addition, it has sometimes been hard to decide whether or not a given case comes within the scope of the present paper.

Under the name "Coloboma nervi Optici," v. Ammon (1858) describes a condition which he found in the early fœtus of the sheep. The fœtal cleft was closed at the level of the sclera, so that from the inner surface the disc appeared to be normal. Further back, however, the cleft in the nerve was open, and formed a large hiatus below. v. Ammon evidently regarded this not as a pathological condition, but as a normal stage in the formation of the nerve, since he goes on to say that later it disappears entirely, or may be observed for a considerable time in the form of a seam. In any case the condition is not that which is at present under consideration, and need not be further mentioned.

Liebreich (1859) seems to have made the earliest pathological examination of a coloboma at the nerve entrance. Having been the first to observe the condition ophthalmoscopically, he afterwards obtained two eyes in which, on section of the globe, it was found associated with coloboma of the iris and choroid. Beneath the nerve, and close to its junction with the sclera, the dural sheath was bulged out into a deep pocket. The opening of the pocket into

the vitreous cavity was spanned by a delicate membrane which originated from the nerve. The nature of this membrane was doubtful; Liebreich believed that it contained no retinal elements, but apparently no microscopical examination was made. The diameter of the opening was about double that of the normal papilla, and the nerve was in the upper part of it, and in contact with its upper margin. Smaller secondary bulgings were present in the floor of the main ectasia. The exact relations of the different tunics at the nerve entrance are not fully described, but it is probable, in view of other cases to be mentioned, that it was a coloboma of the choroid bordering on the nerve.

da Gama Pinto (1884) described a case associated with coloboma of the iris and choroid. The eye was lost in adult life from iridocyclitis. On section an ectatic coloboma of the choroid was found, commencing at the lower papillary margin and ending at the ora serrata. There were cyclitic membranes, and the retina was detached except over the coloboma of the choroid, which showed the usual fusion distortion and thinning of retina and choroid; it is unnecessary to enter into its structure here. The lamina cribrosa was in its normal position. At the termination of the sclerotic and choroid by the lower edge of the papilla, there was a little cavity in the sclera filled with a reticular, richly cellular tissue, similar to the substance filling the coloboma, but separated from it by a narrow bridge of scleral tissue. Its relations to the intervaginal space are not given, but da Gama Pinto speaks of it as a rudimentary coloboma of the optic nerve.

v. Duyse has reported six cases. (1) (1884). A coloboma at the nerve entrance was present in a microphthalmic eye. There was also a coloboma of the choroid. A deep excavation of all the tunics close beneath the nerve is shown. The papilla is in the upper part, and faces downwards, so that ophthalmoscopically it must have appeared much foreshortened. The nerve itself and its sheaths are

represented as normal, the ectasia, therefore, being a coloboma of the choroid beneath the nerve.

(2) (1896). The abnormality of the nerve had not been observed ophthalmoscopically. The left globe was normal in size and shape. Typical colobomata of the iris, ciliary body, lens, and choroid were present. At the nerve entrance there was an excavation measuring 4 mm. vertically, by 3 mm. horizontally. The lower border of the ectasia of the nerve proper, abutted directly on a second excavation, which again was separated by a ridge from the main coloboma of the choroid. Microscopically, the intervaginal space was found to be much enlarged on the nasal side of the nerve. The sclera and choroid were fused and indistinguishable by the side of the nerve, and both were tucked back in the form of a little angle, or kink, into the termination of the widened intervaginal space. The retina also dipped back into the same angle, but was not fused with the underlying structures, nor did it penetrate the sclera at this point. Beneath the nerve there was a deep excavation in the sclera lined with fused retina and choroid, the pigment epithelium stopping short at its edge. Smaller secondary spaces were connected with the main cavity. The borders were sharp and overhung. The nerve sheaths joined the sclera about the middle of the excavation, so that the coloboma was apparently partly in the nerve itself, partly in the choroid beneath it. The sections were cut in a horizontal direction, however, which is not so suitable as the sagittal for the exact determination of the relations of colobomata beneath the nerve.

(3) In the right eye of the same patient there were also colobomata of the iris ciliary body, lens, and choroid. The coloboma of the choroid was very large, engulfing the papilla, but at the nerve entrance there was a separate sharply circumscribed excavation. Microscopically, the lesions were similar to those found in the left eye. There was an extensive defect of the choroid and pigment epithelium by the side of, as well as below, the nerve, while

the retina was imperfectly formed in the same area. Beneath the nerve the sclera was also badly developed, and contained many large cystic spaces. The nerve itself, and its sheaths, seem to have been normal.

(4) (1899). This case is similar to one of mine inasmuch as it occurred in an anencephalic fœtus born at full term. In addition to the coloboma at the nerve entrance slight microphthalmos, a typical coloboma of the iris and choroid, and a persistent hyaloid artery were present in the right eye. In the divided globe a white spot, apparently not exceeding the normal papilla in diameter was observed. Immediately beneath the nerve a little globular cyst 2 mm. in diameter projected; it belonged partly to the nerve, partly to the sclera. Microscopically the central vessels were present in their normal situation. As is usual in anencephaly, no nerve fibres were found in the optic nerve. At the lower border of the papilla a narrow canal penetrated backwards through the point of junction of the sclera and the dural sheath, expanding somewhat after piercing the sclera to form the globular cyst seen macroscopically. The cavity was filled with distorted, but distinguishable retinal elements. The pigment epithelium could be followed to about half its depth, gradually losing its pigment. The choroid stopped abruptly at its edge. The lamina cribrosa ran into the upper lip of the opening, so that the defect was, strictly, outside the proper limits of the nerve; this was also shown by its reaching the exterior of the globe outside the dural sheath.

(5) (1905). In this case the other abnormalities present were a typical coloboma of the iris, a dislocation of the lens and a patch of depigmentation of the choroid below. The eye was microphthalmic. Beneath the nerve there was a projection the size of a pea. A similar pouch had been present in the other eye, but was accidently cut off in excising the globe. The central vessels were absent from the nerve, and ran in the dural sheath beneath it. The papilla faced downwards; the lamina cribrosa was well

developed, and had the same direction. The pouch beneath the nerve was filled with greatly distorted retina. The nerve itself was also defective, a depression being present at its edge (but within the scleral foramen) into which projected a little knuckle of retina which showed "perverse" arrangement of its layers. This was a case of involvement of both the choroid and the nerve.

(6) (1905). The globe was microphthalmic and there was a coloboma of the iris. A large pouch was present beneath the nerve, communicating freely with the interior of the globe. Within it the retina and choroid were much thinned. Its upper border was fused with the external surface of the dural sheath, which had normal relations with the nerve. The nerve itself was properly formed, but wheeled strongly round, so as to face downwards. The case was one of coloboma beneath the nerve, the nerve being altered only secondarily and passively.

A case of Falchi's (1890) is sometimes quoted as an instance of a coloboma at the nerve entrance pathologically examined. Falchi himself uses the term. The description and figure, however, seem to indicate an area of peripapillary choroidal atrophy, not a true coloboma. There was no ectasia of the papilla or sclera. The intervaginal space was much dilated, probably from atrophy of the nerve. The case was one of non-congenital buphthalmos, in a girl aged 16 years.

Manz (1891) describes two cases, one in a human, one in a rabbit's eye. (1) In the human eye the coloboma had been observed ophthalmoscopically. The position of the papilla was occupied by a large white excavated area, varying in depth from −10 D. in the middle, to −13 D. to −14 D. below, the refraction of the fundus being −6 D. The vessels entered in an upward direction from below (Caspar's Group 1) and bent sharply over the edge of the excavation. There were no other abnormalities in the fundus.

In the enucleated eye a slight bulging was visible externally at the site of insertion of the optic nerve.

Microscopically the nerve was found to be present in the upper part of the excavation only. Its relations with the upper margin of the scleral foramen were normal. Below, on the other hand, there was a large scleral defect which was filled in with areolar fibrous tissue containing many cystic spaces; some of these projected into and even through the thinned sclerotic. The areolar tissue presumably represented the supporting tissue of the retina, since it was connected on the one hand with the entering nerve fibres, on the other with the retina. The lower border of the scleral defect was very sharp and overhanging. The choroid could not be traced as such beyond its edge. The central artery and vein were absent from the nerve, but many large vessels were present below, some within and some outside the dural sheath. The sheaths were very thick below, but the inter-vaginal space ended in normal relations with the nerve. The nerve itself was normal. In its main features this was a case of coloboma of the choroid bordering the nerve, as is shown by the normal formation of the lamina cribrosa and the normal relations of the nerve sheaths and inter-vaginal space to the nerve itself. The absence of the central vessels from the nerve, however, and their presence in the sheaths beneath, indicate a defect in the intrusion of mesoblast into the neural part of the cleft. This abnormality did not prevent the formation of the nerve and its sheaths from proceeding normally in other respects.

(2) In Manz's second case a coloboma at the nerve entrance was present in both eyes of an albinotic rabbit. In the right eye it was associated with a posterior polar cataract and a persistent hyaloid artery. In the left there was a large round deep excavation at the site of the papilla. The nerve was in the upper part; the greatest depth of the coloboma was immediately below it, and beneath that again there was a shallower portion separated off by a ridge. On pathological examination a conical ectasia was found below the nerve, and close to it. The anatomical relations were not so clearly shown as in the human globe.

Hess (1892) reports the following case: In the eye of a newly-born anencephalic child there was an oval excavation 2½ mm. deep, by 4 mm. broad, in the situation of the nerve entrance, the nerve running into its posterior part. The edges of the excavation did not unite below, but ran downwards and forwards, forming the thickened borders of a groove or channel which reached as far as the ciliary body. The fixation of the globe was bad, so that Hess does not go into the particulars of the microscopical examination of the nerve. This was evidently a somewhat atypical case of coloboma of the choroid, associated with coloboma at the nerve entrance.

Bock (1893) gives a very full description of three cases: (1) In addition to the coloboma at the nerve entrance there were typical colobomata of the iris, ciliary body and choroid. A bulging was visible in the sclera corresponding to the latter, and at the insertion of the nerve there was a second, separate ectasia. On dividing the globe the papilla was found to be kidney-shaped, with the long diameter horizontal. It was deeply excavated on each side, the two depressions being separated by a ridge which ran downwards towards the edge of the coloboma of the choroid.

Microscopically the nerve was of normal thickness, but inserted higher than usual and in a direction from below upwards. Its relations with the sclera, choroid, and retina were normal above. The central vessels lay beneath the centre, at about the junction of the lower and middle thirds of its diameter. The upper half of the papilla was normally formed; the lower was so much displaced downwards and backwards as practically to have disappeared in the bottom of a pit. The excavation measured about 3 mm. in depth; it was external to the sheaths of the nerve, and caused a projection on the exterior surface of the globe without, however, interfering with the normal formation of the intervaginal space. The coloboma was lined with a tissue more or less resembling retina; the choroid seemed to be completely absent. Beyond the lower edge of the pit a

coloboma of the choroid of the usual type was present. The central vessels entered the nerve much nearer the globe than usual. In the upper half of the papilla their division was normal; in the lower, they ran down the longitudinal ridge already mentioned as separating the coloboma into two parts. In this case the nerve was only secondarily involved in the ectasia. The essential lesion was a coloboma of the choroid beneath its lower edge.

(2) The globe was normal in size. Immediately beneath the nerve there was a sharply localised bulging of the sclera, measuring 7 mm. vertically, 8 mm. horizontally. It was separated from the point of insertion of the nerve by a narrow groove, and the nerve itself was displaced upwards. On section the nerve entrance was visible in the upper part of the coloboma, projecting about 3 mm. above the level of retina. At its lower border there were two funnel-shaped depressions separated by a longitudinal ridge which ran downwards into a coloboma of the choroid. There were also colobomata of the iris ciliary body, lens and vitreous. Microscopically the intervaginal space was much widened below, but became narrower towards the sclera. The upper part of the papilla was swollen and œdematous, the lower consisted of a projecting mass of irregularly reticular dense fibrous tissue, with a thin layer of fibres on the surface seemingly belonging to the retina. At the lower edge of the papilla there was a mass of fibrous tissue containing many vessels. The choroid ended sharply at the edge of the coloboma. No central vessels were found in the nerve. The description in this case is not so clear as in the last, and is not aided by a figure, but it is plain that the chief lesions were not in the nerve, but in the choroid beneath. The absence of the central vessels, however, indicates an abnormality in the closure of the neural as well as of the retinal portion of the fœtal cleft.

(3) The other eye of the same man showed a condition in all respects similar. Both these cases might, perhaps, have been classified clinically as colobomata of the choroid

adjacent to the nerve rather than as colobomata at the nerve entrance.

Ginsberg (1896) describes a case in the eye of a rabbit. There was a coloboma of the iris, and the lens and ciliary body were malformed. The coloboma at the nerve entrance was noticed ophthalmoscopically, but is not described in detail. On enucleation a small bulging of the posterior pole was observed, into which the nerve was inserted. When the globe was divided the papilla was found to be represented by an excavation 2 mm. in diameter, and with sharp overhanging edges. Some atrophic patches were present in the surrounding choroid. From the lower part of the excavation a spindle-shaped mass 2 mm. long projected into the vitreous.

Microscopically there was considerable evidence of choroido-retinitis. The mass projecting from the lower edge of the disc was a fold of retina, deriving blood from large vessels piercing the sclera on the temporal side. The excavation lay wholly within the area of the papilla, which measured about 2 mm.—not much more than its normal diameter. Its posterior surface was lined by the thinned sclera and by the sheaths of the optic nerve. The edge formed by the retina and choroid was prominent. The papilla was composed of supporting tissue irregularly proliferated, the nervous elements being much atrophied. No central vessels were found in the nerve.

Görlitz (1897) made a pathological examination in a case which had been seen ophthalmoscopically. The papilla was much enlarged—about three times its normal diameter. It was brilliantly white downwards, and downwards and outwards; elsewhere yellowish. The borders were sharp and pigmented; the shape obliquely oval. The whole area was excavated—below more than above. The fundus was otherwise normal. The vessels entered with a sharp bend at the edge of the coloboma, except one artery and vein on the nasal side, which arose in the centre.

The globe was not microphthalmic, but the cornea was

small. At the junction of the sclera with the nerve below there was a bulging without sharply delimited anterior or lateral boundaries. Its posterior limits were more plainly marked by a groove. It belonged partly to the nerve sheaths and partly to the sclera. The intervaginal space was widened. On dividing the globe the coloboma was found to measure 3 mm. by 2½ mm., the axes being oblique. With the loupe two areas were visible in the nerve entrance, an upper, fairly flat portion which, however, gradually receded below, and a lower smaller portion which sank steeply into the underlying tissues, and corresponded with the bulging visible through the sclera.

Microscopically the nerve entrance occupied somewhat more than one-half of the excavation at the posterior pole, the remainder being formed by a recess filled with densely intertwined masses of fibrous tissue. The nerve was of normal breadth, the lamina cribrosa prominent and well developed, except for the defect to be mentioned below. The intervaginal space was enlarged above, but in normal relation to the surrounding structures; below it was compressed into a narrow slit owing to the projection of the ectasia, but here also the sheaths were normally developed, and in their normal relations to the lamina cribrosa. Owing to the formation of the ectasia immediately beneath it, the nerve entrance was bent a little round so as to face downwards. There was a defect in the sclera below the papilla, forming the entrance to a recess with a narrow neck and more expanded fundus outside the general surface of the globe. The cavity was filled with a peculiar finely fibrillar tissue representing altered retina. The pigment epithelium ended at the lower edge of the excavation, and the choroid could not be traced further than the same spot. Central vessels were present in the nerve. Somewhat below the centre of the papilla there was a small but almost complete gap in the lamina cribrosa, which was well developed elsewhere. In the upper part of the lamina there was a second, separate defect of the same nature but deeper, extending

through its whole thickness. At the bottom of this defect there was a large empty space.

In this case there were two, probably independent, abnormalities. (1) The ophthalmoscopic picture of coloboma at the nerve entrance was due to a defect, not in the nerve, but in the choroid bordering its lower edge. The case was, therefore, similar to those of Manz, Bock, &c. (2) In addition there were defects within the nerve itself, in the lamina cribrosa. These, however, were filled in with nerve fibres, and were perhaps invisible ophthalmoscopically.

Bach (1898) reports the following cases: (1) Besides the coloboma of the nerve a number of other abnormalities were present—imperfect formation of the cornea, lens, vitreous and retina, persistent papillary membrane, coloboma of the iris, ciliary body, and choroid. In the lower half of the globe the retina did not reach so far forward as above. At the inferior border of the nerve a piece of retina, apparently corresponding to the defective area, was snared off and projected backwards by the side of the nerve. It was connected with the normally placed retina by a narrow canal. The retinal elements present in the recess were distorted and confused in structure. According to the accompanying picture the snared off retina runs backwards in the inner layers of the dural sheath of the nerve, compressing the intervaginal space from without. The nerve itself and the lamina cribrosa seem to be normally formed.

(2) (Bach's Case 5). This case occurred in the eye of a rabbit. There were some minor abnormalities (not colobomata) of the cornea, iris, and ciliary body; persistent hyaloid artery, posterior polar cataract, etc. Beneath the papilla a cavity projected beyond the external surface of the sclera. It was lined with retinal remains, chiefly the supporting tissue. The nerve itself was considerably excavated, and from its inferior edge—at its junction with the ectasia—the persistent hyaloid artery sprang. There were no central vessels in the nerve, the hyaloid artery being supplied

apparently from a vessel in the dural sheath corresponding to a member of the Circle of Zinn. The lamina cribrosa was totally absent. In this case there was a coloboma of the choroid beneath the nerve as well as abnormal formation of the nerve itself.

E. v. Hippel (1898) has reported a case in the eye of a newly-born child. The globe was normal in size and external appearance. Even on dividing it there seemed to be merely a well-marked physiological excavation, so that it was unfortunately omitted to cut serial sections, and the direction of bisection was not noted. All anterior parts of the globe were normal. The pigment epithelium and choroid ended in the usual manner at the edge of the porus opticus, which measured 1 mm. across. Instead of being filled by the entering nerve, however, the porus was occupied by an evagination of retina in the form of an ampullated cyst, whose lumen was connected with the vitreous cavity by a fairly wide canal. The retina within the cyst was much folded, but its structure was little altered. The arrangement of its layers was normal—not "perverse." The cyst projected through the sclera, and was in direct contact with the orbital tissues, without any proper wall. On each side of its neck, little ill-formed pieces of optic nerve were found; the sheaths could not be traced. These two portions of nerve became united above the cyst, but the nerve could not be followed backwards, and was believed by v. Hippel to be absent behind the globe. Central vessels were absent, and the papilla was extremely imperfectly formed. V. Hippel believes that this was a case of coloboma of the nerve itself, not of the choroid beneath it. The intraocular part of the cleft had closed normally, but the neural part had totally failed to come together. Hence the division of the nerve into two parts in its (presumably) lower portion. This explanation is very probably correct, but there seems to be no other quite similar case in the literature.

In Bernheimer's case (1894) the abnormalities of the

eye seemed to be the result of hydrocephalus secondary to a leptomeningitis. The eyes were microphthalmic. In the left a cystic swelling filled with clear fluid was present by the side of the nerve. There were some minor abnormalities in the anterior part of the eye—persistent pupillary membrane, imperfect development of the lens, and of the iris and ciliary body on one side, etc. On the temporal side of the papilla the nerve was encroached upon by an excavation which projected beyond the external surface of the globe. It was lined by a tissue composed of altered nervous elements apparently derived from the retina or papilla, and in its centre there was a cavity communicating by a narrow canal with the interior of the globe. Choroidal pigment could be traced into the recess. The nerve itself was much atrophied; on its temporal side the intervaginal space was compressed and untraceable. The course of the central vessels is not stated. In the right eye the whole posterior part of the globe projected backwards in a long funnel-like ectasia.

The choroid had probably a share in the abnormality in this case since choroidal pigment was present in the cyst. According to the description, the coloboma seems to have been on the temporal side of the nerve, not beneath it, but the inferior relations of the nerve were not minutely studied, since the author was dealing with the connection between microphthalmos and hydrocephalus, not with the subject of coloboma at the nerve entrance.

Elschnig (1900) has examined five cases. Some of them, however, would be classified clinically as colobomata of the choroid touching the nerve, rather than as colobomata at the nerve entrance:—

(1) (Arch. f. Ophth., vol. li, p. 405, Case 3.) Ophthalmoscopically the right disc was larger than the left, and was bounded on the temporal side by a brilliantly white ring, outside which again there was a crescent. The eye was emmetropic and vision was apparently perfect. The white ring was somewhat sunk beneath the level of the surrounding structures, and the retinal vessels made a sharp bend in

crossing it. Microscopically there was a deep excavation in the sclera at its termination on the temporal side of the nerve. The defect cut off the lamina cribrosa from its attachment to the sclera, and passed backwards outside the fundus of the intervaginal space, which retained its normal relations to the nerve. The contents consisted of altered retina, continuous, on the one hand, with the nerve fibres of the papilla; and, on the other hand, with the retina beyond the pocket.

(2) (Arch. f. Ophth., vol. li, 1900, p. 408, Case 4.) In another case, Elschnig describes a small inclusion of rudimentary retinal elements above the lamina cribrosa. The nerve was otherwise normally formed, the connection with the intraocular retina having become lost. Elschnig points out that the case resembles those of Bach and Görlitz; it is also similar to Cases 1 and 2 reported below.

(3) (Arch. f. Ophth., vol. li, 1900, p. 411, Case 5.) In this case there was myopia of 25 D. The papilla was bounded on the temporal side by a white depressed area. Outside this again there was a crescent of the usual type. Microscopically the intervaginal space was greatly widened, both on the nasal and temporal sides, its ending being bridged across by fibrous tissue running inwards from the junction of the sclera with the dural sheath. This bridge was bulged backwards, and was lined on its vitreous surface by spongy fibrous tissue, presumably distorted retina. I have mentioned this case because Elschnig calls it a coloboma at the temporal side of the nerve entrance, and regards it as a congenital abnormality. But, in view of the high degree of myopia, it is not impossible that the state of the nerve was brought about by stretching. Localised bulgings, in addition to the general ectasia of the posterior portion of the globe, are not infrequent in high myopia. The condition known as staphyloma posticum verum is one example, and I have also observed an instance in which the bulging was confined to the nerve entrance. Moreover, in Elschnig's case, according to the accompanying photograph, there seems to have been

no very essential difference between the lesions on the nasal and on the temporal sides of the nerve. The same stretching of the scleral promontory, and distortion of the retina were present, though the nasal excavation was not so steep.

(4) (Arch. f. Ophth., vol. lvi, 1903, p. 49, Case 1.) This eye was obtained *post mortem*. Ophthalmoscopically the papilla was horizontally oval, and was bounded below by a broad, irregular, excavated white area. On enucleation, a bulging and thinning of the sclera were found in the same situation. The nerve was inserted with an upward obliquity, but turned sharply downwards in passing through the scleral opening. On the lower side the sclera formed a rounded knee, over which choroid and retina were absent.

(5) (Arch. f. Ophth., vol. lvi, 1903, p. 52, Case 2.) In this case the corneæ were small and there was a coloboma of the iris. The papilla was oval, horizontally, and bordered below by an irregular crescent-like area. The eye was myopic −10 D. Microscopically the crescent was of the usual myopic type; the choroid and pigment epithelium ceased 1·75 mm. from the nerve entrance below, whereas above they ended only a very short distance from the termination of the sclera. In the scleral promontory below there was a pocket 1·8 mm. deep, filled with nerve fibres and some connective tissue. Vessels entered it from the central retinal system, and it reached the external surface of the globe outside the dural sheath. It was probably independent of the crescent and invisible ophthalmoscopically.

P. Knapp (1901) has published a case in which both eyes of an albinotic rabbit were affected. Ophthalmoscopically the usual deep pit, with the papilla in its upper part, was observed. The excavation formed a little ectasia, visible externally at the lower part of the junction of nerve and sclera. The globe was not microphthalmic. Microscopically the papilla was somewhat cupped, but otherwise normal; the nerve contained a small central vessel. The lower border of the papilla was displaced backwards by the ectasia beneath

it, which measured 6 mm. in diameter by 3 mm. in depth. Its walls consisted of much thinned sclera (1/10th normal thickness). The choroid could only be traced a little way beyond the edge of the excavation. The retina and the retinal pigment, on the other hand, were not interrupted, though the former was much atrophied, especially in its nuclear layers. The condition was practically the same in each eye. This is a good example of a coloboma beneath the nerve, the nerve itself being normally formed and sharing only passively in the ectasia of the coloboma.

Tschemolossow (1901) has reported a case associated with microphthalmos and coloboma of the iris and choroid. The coloboma at the nerve entrance was distinct from the defect of the choroid. At the lower edge of the disc there was an excavation with overhanging edges. Clumps of pigment were present in the adjacent sclera and choroid. In the floor of the pit, retinal elements were found. I have been able to consult this paper in a short abstract only; it is impossible to say exactly what relations obtained, and whether the case was one of defect in the nerve itself or in the choroid beneath it.

Monthus and Opin (1903) have reported an instance which more nearly resembles Cases 1 and 2 of the present paper than any other in literature. It occurred in a child which died shortly after birth at full term. In addition to various other abnormalities in other parts of the body, the eyes were microphthalmic. At the upper part of the right nerve there was a cleft or cavity which separated the nerve substance from the pial sheath. It occupied about one-third of the circumference of the nerve. The inner surface of the pial sheath, where it lined the cavity, was clothed with epithelial cells, and in many places these cells contained pigment exactly similar to that normally found in the pigment epithelium. It is not clear whether the cleft communicated with the interior of the globe. The explanation of this condition will be discussed later.

Gilbert (1907) describes two cases in which aplasia of the

lower part of the nerve was associated with a coloboma of the choroid. In the first the nerve was grooved below, so that its transverse section was kidney-shaped. The intervaginal space commenced further back than on the upper side. The nerve and its sheaths were otherwise normally formed, and the central vessels were contained within the nerve. The second case was similar, but without grooving of the nerve. Gilbert ascribes the condition to a congenital defect from undue persistence of the mesoblast. In all such cases, associated with ectatic colobomata of the choroid, it is obviously well-nigh impossible to disentangle an aplasia due to defective formation, from an atrophy caused by loss of nerve fibres owing to destruction of the retina in the colobomatous area. Atrophy and grooving of the lower part of the nerve may also be caused by direct pressure of the ectasia.

Personal Observations.

Case 1.—The globes in this and the following case were derived from an anencephalic child which was born at full term and lived 36 hours. After death the whole child was placed in 10-per-cent. formalin. I am greatly indebted to Dr. Gordon Holmes, who obtained the eyes for me.

Externally the globes appeared normal except for the thinness of the optic nerves. They measured 18 mm. anteroposteriorly. The muscles were either so poorly developed or had been cut off so closely that it was impossible to tell which eye was right and which left; for the same reason the upper could not be distinguished from the lower aspect. I have presumed that the defects were at the inferior edge of the papilla, not above. The globes were divided sagittally on the temporal side of the nerve. On section, the papilla did not appear to be enlarged or greatly excavated, while all other intraocular structures were normal. The condition of the nerve was found accidentally on microscopical examination of one of the eyes; this globe had not been cut serially.

The other globe was then cut in serial section ; the description below refers to this second globe.

Microscopical Examination (Pl. VI, Fig. 1).

The cornea, iris, lens, and ciliary body are all normal. In the retina (*r*) the nerve fibre layer is totally absent. There is a layer of cells corresponding in position to the ganglion cells, but fixation is not sufficiently good to make it quite certain whether these are true nerve cells or nuclei belonging to the neuroglial tissues. The retinal vessels are in this layer, and are separated from the vitreous by the limitans interna alone. All other layers of the retina are normal; the rods and cones are present, but have suffered in fixation. The choroid is normal ; corresponding with the age of the child, it contains no stroma pigment.

At the (presumably) lower edge of the nerve the defect shown in Pl. VI is present. It consists essentially of a deep pocket filled with altered retinal elements (E). It is important to note its exact relations. It passes backwards at the point of junction of the lamina cribrosa (*l*) with the scleral promontory (*s*) at the lower edge of the nerve, cutting off the attachment of the lamina to the sclera, and causing a defect in the former. The lower edge of the lamina is slightly tilted backwards. It next comes into relation with the *inner* (neural) aspect of the intervaginal space (*v*), which undergoes compression and some displacement from the neural side. It then passes backwards into the lower part of the nerve, where it hollows out for itself a large irregular cavity, terminating finally in a blind end at a distance of 1·25 mm. above the level of the choroid. So far as it extends, practically the whole inferior half of the nerve is destroyed by it. The defect is entirely within the limits of the nerve itself, the adjacent choroid (*ch*) and sclera (*s*) taking no part in its formation. In agreement with this, the diameter of the scleral and choroidal openings at the nerve entrance is little, if at all, greater than normal ; it measures 1·25 mm. at its widest part.

The entrance to the pit forms a comparatively broad channel which expands somewhat, deeper in the nerve. At the edge of the canal of entrance the choroid comes to an abrupt end in an overhanging spur (*ch*). There is not the least trace of atrophy, thinning, or inflammation up to its point of termination. The pigment epithelium runs over the spur of choroid and lines the excavation, as will be described presently. The opposite border of the canal of entrance is formed by the tissues of the lamina cribrosa (*l*), clothed with a quantity of neural supporting tissue. The sclera (*s*) like the choroid is perfectly normal up to the wall of the defect, where it ends abruptly. Its outer layers pass into the dural sheath of the nerve (*d*) in the normal manner, and its relations to the intervaginal space (*v*) are normal, except that the space ends sooner than on the opposite side, owing to its compression by the pit in the nerve. The upper relations of the nerve are normal.

The contents of the excavation consist of the distorted retina usually found in colobomata (E). It is composed of a loose cellular fibrous tissue probably representing the neuroglial supporting tissue rather than the nervous elements. The nuclear layers cannot be traced so that it is impossible to say whether the arrangement is normal or "perverse." At the mouth of the pit this tissue runs directly into the retina on the one hand, and into the tissues of the papilla on the other. In the deeper parts of the nerve similar loose fibrous tissue is present, but here it contains many cystic spaces. Isolated patches of similar tissue are found in the opposite half of the nerve (*p*). They have no connection through the lamina with the tissues of the papilla, or, within the nerve, with the main defect.

The whole of the excavation down to its smallest recesses is lined with a layer derived from the pigment epithelium, which is only defective in small areas here and there. At the mouth of the pit this layer runs directly into the pigment epithelium of the choroid. Tracing it backwards, it retains its pigment for a short distance and then gradually

loses it, leaving a lining of unpigmented cells. Deeper in the nerve it again acquires pigment in patches (*a*) alternating with unpigmented areas. In the isolated patches on the opposite side of the nerve pigmented areas are also recognisable.

The mouth of the excavation does not occupy the whole of the inferior portion of the nerve, but probably about half its breadth. It is also situated towards the temporal side, so that sections passing through the middle of the nerve entrance do not touch it. In the absence of any nerve fibres one can scarcely speak of a papilla, but in sections which pass through the excavation a mass of tissue similar to that found within the pit (neuroglial tissue) lies in front of the lamina cribrosa, and in some sections projects a certain distance into the vitreous. In sections which pass by the side of the pit, through the centre of the nerve entrance, there is no such tissue; the lamina cribrosa is here practically laid bare. There is, therefore, a steep edged cupping of the whole nerve entrance (in its centre; not shown in Pl. VI), but it does not resemble a glaucomatous cup, inasmuch as the lamina cribrosa, though obliquely set, is not bowed backwards. Except for the defect where the canal of the excavation penetrates it, the lamina cribrosa is perfectly well developed.

The nerve itself consists of the supporting framework only, without, so far as can be made out, any trace of nerve fibres. It is exceedingly small and shrunken, and between the coarser trabeculæ there are collapsed masses of finely granular material. These evidently represent the ultimate neuroglial supporting tissue of the nerve. In places the sheaths are not properly differentiated, dura, arachnoid, and pia being indistinguishably fused. Elsewhere again, and especially near the posterior surface of the sclera, they are quite well formed.

The central vessels enter the nerve 3·75 mm. behind the level of the choroid, and pass into the centre in the normal manner. In the posterior part of the nerve they remain in

this situation, but on reaching the colobomatous area they undergo displacement towards the upper part of the nerve, so that they enter the globe immediately beneath the upper border of the papilla (*rv*). No vessels are present within the abnormal pocket.

Case 2 (Pl. VII, Fig. 2).

The above description might be applied almost word for word to the conditions found in the second eye, so that it is sufficient to indicate points of difference. There is evidence of inflammation of the choroid in the form of a broad patch of round cell infiltration in the region of the ora serrata. In the neighbourhood of the papilla, however, the choroid is quite normal. The defect beneath the nerve (Pl. VII, Fig. 2) is less complex in structure, the distinction between the pigment epithelial lining and the contained retina being more evident. It encroaches a little more on the scleral promontory, but comes into relation with the intervaginal space on its neural side in the same manner as in the other eye. It does not reach so deeply into the nerve—very little above the lamina cribrosa. The complex system of cavities found in the deeper parts of the nerve of the other eye is therefore absent here. An important difference between the two eyes is found on the side opposite the main defect. In the first eye the relations were normal in this situation. In the second the pigment epithelium runs over the termination of the choroid and forms a little dip into the tissues by the side of the nerve entrance. Some retinal elements are present in the minute pocket so formed. It is cut peripherally and no more than indicated in Pl. VII, Fig. 2. This is, in fact, a rudimentary example of the same conditions as are found at the lower edge of the nerve. In the upper half of the nerve also there are large retinal inclusions (*p*), commencing at the posterior level of the lamina cribrosa and extending about 0·85 mm. above that point. These are much more prominent than in the other eye. So far as the sections show, there is no connection

between them and the surface of the papilla or the excavation on the inferior side of the nerve, but, as stated, the series is incomplete in this eye. The retinal elements within these inclusions are much better distinguishable, and in places a limiting membrane lined with layers of nuclei is present (Pl. VII, Fig. 2). These evidently represent the limitans externa and the outer nuclear layer. Distinguishable rods and cones are not present. Cystic spaces are not so plentiful within the retinal inclusions.

Case 3 (Pl. VII, Fig. 3, and Text-figure A).

The following case, in which I had the privilege of making the pathological examination of the eye, has already been reported by Mr. J. Herbert Parsons and myself (Brain., Pt. CXIV, 1906). For the details of the rare abnormalities which were found in the brain and eyeball I must refer readers to that paper, confining myself here to the condition of the optic nerve entrance.

The specimen was derived from a child ten months old. There was a large meningo-encephalocele of the right orbit which displaced the eye downwards and outwards. A very deep coloboma at the nerve entrance was visible ophthalmoscopically, as well as a macular coloboma and an excavation on the nasal side of the papilla. The globe was small, measuring antero-posteriorly 18 mm., transversely 19 mm., and vertically 16·5 mm. At the junction of the nerve with the sclera there were two cystic swellings. One, on the nasal side, formed an almost perfect hemisphere 5 mm. in diameter. The other was situated further back, and appeared to hollow out the nerve itself; its greatest transverse diameter was 6·5 mm., and from its posterior pole a thin but apparently normally formed nerve projected, The two cysts were separated by a constricted neck, and both were very thin-walled and translucent.

On dividing the globe the most evident abnormality is an exceedingly deep pit or funnel in the position of the optic

disc. The retina is not defective, and on all sides reaches up to and passes over into the excavation. The edge is very sharp and prominent on the temporal side, more shelving on the nasal. The retinal vessels emerge chiefly on the nasal side in several isolated trunks, and the papilla seems to be stretched out over the wall on this side. The cyst ends far back in the nerve in a rounded fundus, but as only a small circular piece was removed from the upper part of the globe this could not be very closely examined. This excavation in the nerve corresponds with the posterior of the two cystic swellings visible externally; the anterior is an ectasia of all the tunics on the nasal side of the nerve. (For details see *loc. cit.*) All the anterior parts of the eye are normal. The globe was fixed in formalin and cut in horizontal serial sections.

Microscopical Examination (Pl. VII, Fig. 3, and Text-figure A).—The abnormality is essentially a deep excavation in the temporal portion of the nerve entrance, the nerve itself being spread out over the wall on the nasal side. The sclera and choroid at each edge of the pit (*t* and *n*) come to an end in normal relations to one another, and to the termination of the intervaginal space (*v*). This is best seen on the temporal side (*t*) because on the nasal (*n*), the intervaginal space has been laid open owing to excision of the dural sheath in removing the globe. On the temporal side then, the sclera ends in a promontory a short distance to the inner side of the termination of the intervaginal space (*v*). The choroid ends in the same position, and the retina (*r*) a little further in, being dragged round into the excavation for a short distance.

On the nasal side the papilla (*p*) is present. It is greatly elongated in a vertical direction, and so much slewed round by the giving way of the tissues on the temporal side, that its direction is at right angles to the normal, and it faces temporally towards the opposite edge of the excavation. The lamina cribrosa (*l*) is well formed; on the nasal side it springs in the usual manner from the scleral promontory; on

the opposite side it runs into a mass of fibrous tissue (*f*), which fills the bottom of the excavation. In the absence of the intervaginal space on the nasal side the attachment of the lamina to the sclera furnishes an important point of orientation, and serves to demonstrate that the sclera and choroid end, as on the temporal side, in normal relations to one another. The lamina cribrosa is not stretched in a transverse direction, in other words the area of the entering nerve is not expanded transversely. Hence it occupies only a very small portion of the wall of the excavation in horizontal sections. It will be noticed from the above description that there is nothing of the nature of a glaucoma cup; the lamina is not bowed backwards, but displaced bodily.

The floor of the excavation is formed chiefly of dense fibrous tissue (*f*). On the temporal side it passes into the scleral promontory (*t*), on the nasal, as mentioned above, it receives the attachment of the temporal end of the lamina cribrosa. Tracing it in greater detail from the temporal towards the nasal side, its structure and relations are as follows:—Taking its external relations, it first comes into connection with the intervaginal space of the temporal side (*v*). The space lies on its outer wall and is somewhat compressed and pushed aside, but quite distinguishable. Further back the dura has been cut away in excising the globe, so that the intervaginal space ceases to exist as such, but the arachnoid sheath (*a*) still continues to line the outer surface of the excavation. A short distance to the temporal side of the deepest part it comes into relation with the nerve (*o'*), which lines its outer surface as far as the lamina cribrosa (*l*). With regard to the internal relations of the fibrous floor of the pit, it is first in contact with the retina (*r*), which is pulled over the temporal edge of the excavation. At first, the retina is well formed, and its layers are distinguishable, but very soon it becomes converted into an indefinite fibrous tissue, probably consisting chiefly of the supporting fibres. This distorted retina hollows out a

place for itself in the underlying fibrous tissue (*r'*). In all sections, however, a few fibrous strands separate it from the intervaginal space, which at this point lines the outer surface of the cyst. Further back, retinal elements totally disappear, and the floor is lined by dense fibrous tissue which may, or may not, represent compressed retinal supporting tissue. This state of affairs is present as far as the temporal attachment of the lamina cribrosa.

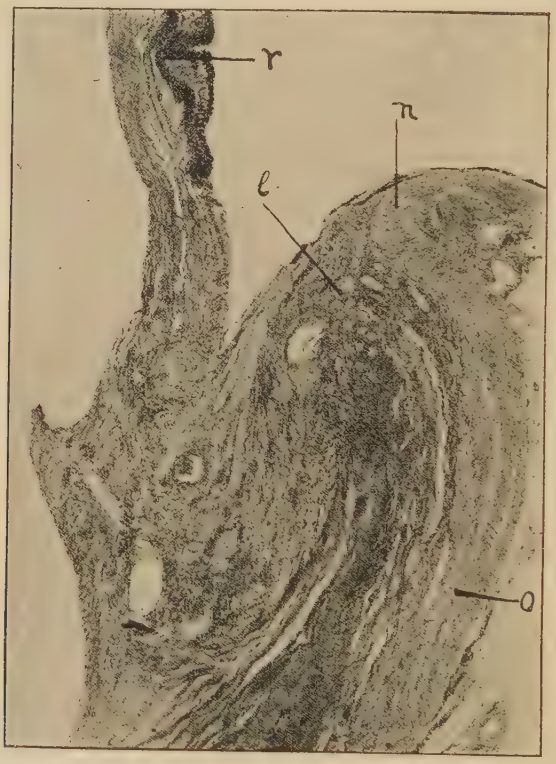

Fig. A.—The Nerve Entrance. *n*, termination of sclera on nasal side; *l*, lamina cribrosa; *r*, retina; *o*, optic nerve.
(From Brain, p. 114, 1906).

Having traced the relations of the fibrous tissue forming the major part of the floor of the excavation, I now return to

the nerve and nerve entrance (Figs. 3 and A). During the formation of the ectasia on the temporal side, the nerve has become spread out over its wall. Hence, in the sections the whole of the nasal and posterior outer surface of the cyst, as well as part of the temporal, is lined by a layer of nerve fibres (*o*). These are closely in contact with the fibrous tissue lining the bottom of the cyst; there is no space which might be supposed to represent the intervaginal space. In consequence of the displacement of the lamina, and of the distortion of the papilla, the nerve fibres have a very tortuous course to pursue in order to reach the retina. They arrive at the lamina at an acute angle to its posterior surface, so that they must bend round very sharply to pass through its meshes. Immediately after passing through, they again turn downwards into the fibrous lining of the excavation, which splits to receive them. Finally, they sweep round in a curve of small radius (*p*), and enter the nasal retina. Their course from the posterior surface of the lamina to their entrance into the retina thus forms a complete S. No fibres reach the retina on the temporal side across the floor of the excavation; if any gain that side they must pass round the rim. But since the nerve fibre layer is very poorly developed or absent on the outer side, while it is prominent nasally, it is probable that very few fibres reach the temporal retina.

In the spur of nerve which lines a small portion of the temporal side of the excavation (*o'*), many fibres are cut transversely. These do not enter the globe on the temporal side, however. They are all mounting over the wall of the cyst to reach the lamina cribrosa at a higher or lower point; this is the necessary consequence of the spreading out of the nerve due to the lateral encroachment of the excavation.

Behind the cyst the nerve passes straight backwards. So far as one can judge, it is little if at all atrophic. Its thinness on the side of the cyst is therefore due, not to atrophy, but to its distribution over a larger area. Central vessels are present in the nerve. Their point of entrance cannot be

determined with exactitude, but is somewhere behind the fundus of the excavation. At the lamina cribrosa they take the same tortuous course as the nerve fibres. Vessels are present in the retina on the temporal as well as on the nasal side; they must have reached that situation by passing round the edge of the pit, since they do not cross its floor.

Review of the Literature and Remarks.

In admitting cases to the abstract on p. 180, *et seq.*, or excluding them from it, I have been guided chiefly by their resemblance or otherwise to the ophthalmoscopic condition, "coloboma of the optic nerve," as defined at the beginning of this paper. In a few instances it has been necessary to mention abnormalities which would have formed a different clinical picture, or which would have been invisible with the ophthalmoscope (v. Ammon, Bach, v. Hippel, etc.). A review of the literature will be found to justify a statement already made by v. Hippel, that the lesions even in cases which closely resemble each other clinically, present no uniformity microscopically. For this reason, v. Hippel has very properly suggested that the term "coloboma of the nerve" should be replaced by the term "coloboma at the nerve entrance." This would serve to emphasize the important fact that, except within narrow limits, it is impossible from the ophthalmoscopic appearances to predict what the microscope will reveal.

From the pathological standpoint, the cases might be classified in the following three divisions:—

(1) The lesion is a coloboma of the choroid beneath the nerve, the nerve itself being normally formed, and sharing only passively in the deformity. To this category belong the cases of Liebreich, da Gama Pinto, v. Duyse (1, 3, 4, 6), Manz (2), Bock (1), Görlitz, Bach, Knapp (1, 2), Elschnig (4, 5); fourteen cases in all.

(2) The lesion is a coloboma of the choroid and nerve. To this category belong the cases of v. Duyse (2, 5), Manz (1),

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Hess (?), Bock (2, 3), Bach (2), Bernheimer, Elschnig (1); nine cases.

(3) The lesion is a coloboma of the nerve alone, the adjacent choroid being normal. To this category belong the cases of Ginsberg, Görlitz, v. Hippel, Elschnig (3 ?); four cases.

It should be mentioned, however, that the classification of some of these cases is doubtful. For instance, it is uncertain whether those of da Gama Pinto, Hess, and Bernheimer are colobomata of the nerve or of the choroid beneath it, and whether Elschnig's Case 3 was a true congenital abnormality. As stated above, v. Ammon's case probably belonged to a different category, and in Tschemolossow's sufficient details are not available in the abstract to which I had access to justify its classification. Görlitz's case appears in Groups 1 and 3, because there seemed to be two distinct abnormalities.

Even with the most exact description, classification becomes difficult when the coloboma is large and much expanded. The relations of surrounding structures are obscured, and the nerve is mal-directed and distorted. In this case attention should be directed to two points—the relations of the intervaginal space to the ectasia, and whether or not the nerve contains central vessels. Normally the intervaginal space is bounded on its inner (neural) aspect by the pial sheath, and comes to a blind end close under the scleral promontory by the side of the nerve entrance. It is evident that any excavation or pit which comes into relationship with it from its inner side cannot be a coloboma of the choroid, and must, therefore, be a coloboma of the nerve. On the other hand, an ectasia which comes in contact with it from the outer (dural) side will almost certainly be a coloboma of the choroid adjacent to the nerve, though it is conceivable that in exceptional instances a coloboma in the periphery of the papilla might pierce the scleral promontory obliquely, and reach the exterior of the globe outside the dural sheath.

With regard to the central vessels, their absence from the nerve amounts to proof of an atypical development. It does not establish a diagnosis of coloboma in the strictest sense, since the fœtal cleft may have closed quite normally, but without including the vessels, and the nerve itself may have been in every other respect properly developed. On the other hand, the presence of the vessels within the nerve does not exclude the co-existence of a coloboma, since the mesoblast containing the vessels may have entered in the usual manner, although the fœtal cleft has failed to close—or, indeed, the persistence of an abnormal amount of mesoblast may be the very cause of the failure in the closure. The co-existence of a coloboma of the nerve with normal central vessels is illustrated in all my cases.

In most instances attention to these points will serve to distinguish between a coloboma of the nerve and a coloboma of the adjacent choroid. In some respects, however, it is a moot point whether it is of much practical importance to maintain the distinction. It has already been remarked that it does not correspond with appearances separable by means of the ophthalmoscope. Pathologically also, the gap between coloboma of the adjacent choroid and coloboma of the nerve is bridged by coloboma involving both these structures. In the vast majority of defects at the nerve entrance, as in colobomata elsewhere, the rule holds that the abnormality occurs in the line of the fœtal cleft, but the neural and intraocular portions of the cleft are directly continuous with one another, and any hard and fast distinction between defects of one or the other portion, though for certain purposes convenient, is to some extent artificial.

Groups 1 *and* 2: *Coloboma of the Adjacent Choroid and Coloboma of the Choroid and Nerve.*

In attempting to sketch the general features of coloboma at the nerve entrance, and to correlate them with the ophthalmoscopic appearances, it will be best to take

together the first two groups mentioned above. The minimal degree of this form of coloboma seems to be a small snared-off pocket containing distorted retinal elements (cases of da Gama Pinto, Bach, 1, Elschnig, 2, 5). It projects into the underlying sclera, nerve sheaths, or nerve according to its situation, and is usually connected with the intraocular retina by a narrow canal. Cystic spaces are frequently present in its depths. Ophthalmoscopically this form is, perhaps, sometimes invisible, or it may reveal itself as a small irregularity in the contour of the disc, or as a narrow white area like an enlarged scleral ring or atypical crescent. As opposed to an ordinary crescent, its floor is frequently slightly sunken, so that the central vessels make a little bend in crossing it (Elschnig).

In higher degrees of the defect, ectasia of the colobomatous area is added, while the defect itself is probably more extensive. Since the coloboma is practically always at the lower border of the nerve entrance, the inferior attachment of the lamina cribrosa sinks backwards, while the upper retains its normal relations. In consequence the lamina becomes swung round, so as to be directed from above downwards and backwards. The papilla of course shares in the displacement, and looks more or less downwards. In extreme degrees the lamina will be directed almost horizontally backwards, and the papilla will face straight downwards towards the opposite wall of the ectasia. Whether the coloboma is in the lower part of the papilla or in the choroid beneath it, the nerve will be impinged upon by the excavation from the lower side, and will become stretched out over the upper wall of the cyst. Viewed with the ophthalmoscope the appearance produced will be that of a deep pit in the position of the nerve entrance, possibly many times its normal diameter. It will have a brilliantly white floor, since even if the choroid were originally present it will probably have become atrophied. The excavation will usually not be equally expanded in all directions, so that secondary depressions will be visible

in its fundus. At the upper part a greatly foreshortened view may be obtained of the displaced papilla, in the form of an indefinite pink area. If the excavation be very steep however, or if the edges be overhung, the papilla may be invisible. Especially will this be the case if the vessels do not enter there.

With regard to the central vessels several arrangements are possible according to Caspar's classification (p. 178), and some of these have been proved microscopically. They may enter in their normal position in the centre of the papilla (v. Duyse, 3, 4, Bock, 1, Görlitz, Knapp, Case 3 above). In this case they will be visible in the pink area in the upper part of the coloboma, and Caspar's second group will be reproduced. The branches destined for the upper part of the retina will reach their distribution comparatively easily; those destined for the lower part must either cross or skirt the coloboma. In the former case they will come over the lower edge with a sharp kink.

If the central vessels fail to enter the nerve they belong to exactly the same category as cilio-retinal twigs of the ordinary type—branches of the ophthalmic artery (or its offshoots) which pierce the sclera by the side of the nerve. Although not within the nerve, however, the central vessels may still form the chief or only blood supply of the retina. In this case they will enter in a group outside the papilla, in the lower part of the coloboma, and Caspar's first group will be reproduced (v. Duyse, 5, Manz). On the other hand, no special set of vessels may be differentiated for the supply of the retina, which will then receive blood from extraneural, *i.e.*, cilio-retinal, vessels, which enter all round the circumference of the coloboma. This will give Caspar's third group. This distribution has not been specifically proved microscopically, but was probably present in some of the cases in which the central vessels are not mentioned as found beneath the nerve, though they were absent from its interior. In this group the ophthalmoscopic appearances simulate those found in animals whose retina is supplied by

cilio-retinal vessels alone.* But the resemblance is merely superficial, since in the human eye it is not an otherwise normal, but apparently always a mal-formed disc, which is so supplied.

In the beginning of this paper the clinical distinction between "coloboma of the nerve" and "coloboma of the choroid adjacent to the nerve" was mentioned. It is evident from the cases already quoted that this distinction, though quite reasonable on clinical grounds, cannot be maintained on pathological. Some, indeed most, of the "colobomata of the nerve" *are* colobomata of the choroid adjacent to the nerve. It is probable that the ophthalmoscopical difference depends chiefly on the amount of ectasia. If this is absent or slight the papilla will be seen in surface view, and will be clearly demarcated from the coloboma; if it is great the papilla will be much foreshortened, and its boundaries will not be sharply delimited, so that it will seem to be replaced by the excavation.

Group 3: Coloboma Confined to the Nerve.

With regard to the much rarer form of defect, coloboma confined to the nerve itself, only three unequivocal examples are to be found in literature (Ginsberg, Görlitz, and v. Hippel). The reasons for questioning Elschnig's case have already been given (p. 193). Even these three do not present much in common; Ginsberg's occurred in the eye of a rabbit, and was in the form of a deep excavation; in Görlitz's there was no excavation, but simply a defect in the lamina cribrosa, filled up with nervous tissue and probably invisible ophthalmoscopically; v. Hippel's was a very peculiar case, for the details of which I must refer to p. 191. What ophthalmoscopic picture would have corresponded with it is very doubtful.

The three cases which I have described above were all

* *E.g.*, seals, certain squirrels, etc. See G. Lindsay Johnson, Contribution to the Comparative Anatomy of the Mammalian Eye, etc., Phil. Trans., 1900.

true colobomata of the nerve alone, the adjacent choroid being quite normal. In Cases 1 and 2 this is evident at a glance (see Figs. 1 and 2). The nerve entrance is practically of normal dimensions, and all the abnormalities are within its boundaries. The pocket of distorted retina passes through the lamina cribrosa and hollows out the nerve, leaving the terminations of the choroid, sclera, and intervaginal space intact, and in their normal relations to one another.

In Case 3 it is not so evident at first sight that the nerve alone is involved. The distance between the ending of the choroid on each side is many times the diameter of the normal disc. Yet careful consideration will show that all three cases are in reality the same. The only essential difference is that ectasis has been superadded in the third. If the defect shown in Figs. 1 and 2 be imagined to become ectatic under the intraocular pressure, the conditions found in Case 3 would be almost exactly reproduced. The lower end of the lamina would be swung backwards, and the papilla would come to occupy the upper part of the excavation, and would face downwards. The nerve would be encroached upon from the lower side, and would be spread out over the upper wall. The connection between the entering nerve fibres and the retina in the lower part of the globe would become lost owing to atrophy in the bottom of the pit. But in spite of all these changes, and however great the depth of the excavation, or the diameter of its mouth, the intervaginal space would still be on its exterior wall, and the endings of the choroid and sclera at its edge would still be in normal relations to one another and to the termination of that space. This corresponds almost exactly with the conditions found in Case 3, if temporal and nasal be substituted for lower and upper, as the ectasia was on the temporal side, not below. Since, however, there was a defect in the lamina in Cases 1 and 2, and apparently none in Case 3, it is probable that the defect was a little more towards the periphery of the nerve in the last. It was presumably the point of attachment of the lamina to the scleral promontory which gave

way. Hence the ectasia, although still internal to the intervaginal space, caused no gap in the lamina, but displaced it bodily.

Case 3 was of extreme rarity in another respect. The coloboma was not below, but on the temporal side of the nerve; the papilla was therefore displaced to the nasal side and the disc faced temporalwards. That this was neither an error in orientation nor due to rotation of the globe by the pressure of the encephalocele, was proved by the presence of an imperfect, but quite recognisable fovea in the usual position. There can be no doubt that the fœtal cleft had little or nothing to do with the abnormalities in this case. Their atypical situation and unusual characters were probably determined by direct interference with the growth of the globe owing to the encroachment of the encephalocele. This applies to the macular coloboma, and to the ectasia on the nasal side, as well as to the coloboma at the nerve entrance.

The combination of coloboma at the nerve entrance with anencephaly, which was present in Cases 1 and 2, is rare but not unknown (see v. Duyse, 4, and Hess). In my own cases the usual features of anencephaly, which have been frequently described, were shown. The retina was well formed except that the nerve fibre layer was absent; even the ganglion cell layer seemed to be represented. At the nerve entrance and in the nerve the mesoblastic framework had been made ready, but no nerve fibres had entered it. The absence of cupping proved that it was strong enough to resist the intraocular pressure. It is not impossible that this condition of the nerve entrance was responsible for the coloboma. There may have been too much room in the scleral foramen, so that a portion of the secondary optic vesicle became stuffed into it. This is suggested not only by the appearance of the main pocket, but also by the little depression which the pigment epithelium and retina form at the opposite side of the disc. The little islands of retinal tissue which were found quite isolated, and far up the nerve, might be derived from

small pieces of the secondary optic vesicle snared off at an early period of development.

Another possible explanation which has much in its favour, is practically the same as that given by Monthus and Opin for their case. In normal circumstances the outer layer of the intraocular portion of the optic vesicle forms the pigment epithelium, and the inner the retina. But in the neural portion these two layers do not form different structures; both alike form an epiblastic framework, guiding the nerve fibres in their growth from the retina. In the fully formed nerve they are probably represented by the ultimate neuroglial supporting tissue among the nerve fibres. It may happen, however, as a congenital abnormality, that a portion of the neural part of the vesicle *does* become differentiated more highly to form elements which ought properly to be found only within the globe. If this occurs there will be a lining of pigment epithelium on the inner surface of the pial sheath (outer layer of optic vesicle), and malformed retinal elements deeper in the nerve (inner layer of optic vesicle). This would account exceedingly well for various points in Cases 1 and 2, *e.g.*, the fact that although a quantity of retina and pigment epithelium is present in the nerve, there is no defect in these structures within the globe. It also accounts well for the isolated little patches of pigment epithelium and retinal elements far up the nerve on the side opposite the main defect. It will be noticed that such an abnormality might occur in any part of the nerve, and has no necessary connection with the fœtal cleft. In the case of Monthus and Opin it was situated, in fact, at the superior border. The importance of this will be mentioned later in connection with the subject of crater-like hole in the papilla.

As bearing on this subject, it should also be noted that even within the globe the differentiation of the outer layer of the optic vesicle into pigment epithelium, and of the inner into retina is not always strictly adhered to. I have observed a case of cyclops in which the pigment epithelium lining a

considerable portion of the choroid first lost its pigment, and then became thickened to form a retinal normal in appearance, but "perverse" or inverted in the arrangement of its layers—with the rods and cones pointing towards the interior of the globe. The area was not colobomatous, and over the whole of it a normal retina was also present, with the rods and cones pointing externally. The obvious explanation of this condition of affairs is that a portion of the outer layer of the optic vesicle formed retina instead of pigment epithelium. A moment's consideration of the manner in which the inner layer becomes continuous with the outer layer by reflexion at the anterior edge of the vesicle will show that if retina be formed from the outer layer it *must* be inverted. Possibly the same explanation holds good for the commoner occurrence of inverted retina in colobomata.

Ophthalmoscopical Appearances. "Crater-like Hole" of the Optic Disc.

With regard to the ophthalmoscopical appearances, Case 3 was seen, Cases 1 and 2 not. In Case 3 the examination was very difficult from the age of the patient, the extreme proptosis and outward displacement of the eye, and the presence of the other abnormalities. The coloboma at the nerve entrance was noted, however, and seemed to be of the usual type. It will be seen from the pathological examination that the case affords the fullest confirmation of v. Hippel's statement that one cannot tell from the ophthalmoscopical appearances what will be the pathological anatomy. It would have been wholly impossible to say clinically whether this was an instance of coloboma of the nerve itself or of the adjacent choroid. The disc was replaced by a deep excavation many times its normal size, and with a greatly foreshortened view of the papilla at one side—in this case the nasal instead of the upper side; it has already been shown that exactly the same appearance is usually due to an ectatic coloboma of the choroid adjoining the disc.

The ophthalmoscopical appearance in Cases 1 and 2 can only be guessed. It is in the highest degree probable, however, that the condition known as "crater-like hole in the disc" depends on a lesion of this nature. This abnormality, little regarded even in special monographs, is characterised by the presence of a deep pit close to, or touching the edge of the papilla. Some 15 or 16 examples are to be found in the literature. In a large majority (10) of the cases the abnormality has been in the temporal half of the papilla, usually in the lower quadrant, more rarely on the horizontal meridian, and apparently never in the upper quadrant. It has occasionally been observed in the inner half of the disc (Wiethe, Remak), and once at its lower edge (Wiethe). In Wiethe's case two pits were present in the same disc, one on the nasal side, one below. The hole never encroaches beyond the edge of the papilla, which is usually normal in shape, or if there be any slight elongation of one or other axis, a regularly oval papilla results such as might well be found in a normal eye. Occasionally, however, the disc is eccentrically expanded and enlarged in the situation of the abnormality (Thomson and Ballantyne). The shape of the opening is usually oval, the long axis being concentric with the disc margin; the extremities of the oval may be pointed (Szili, 2); sometimes it is circular (Szili, 1, Makrocki), or triangular with the angles rounded (Lichtenstein), or slit-like (Remak). It may be within a large physiological cup or connected with the cup by a groove (Remak). The central vessels have usually their normal arrangement; sometimes their point of entrance is a little displaced towards the nasal side; if one of the retinal vessels happens to cross the pit, it very often dives into it, to reappear on the farther side (Wiethe, Stood); occasionally a vessel emerges from it (or in the case of a vein disappears into it) without connection on the papilla with the other central vessels (Wiethe, Peltesohn, Dyckmeester). The edge is always very sharp, and the cavity is commonly directed straight backwards into the nerve; sometimes, however, it is very oblique, so that the deeper

parts are hidden (Remak), or it slopes gradually for a certain distance and then passes straight backwards (Gunn). The depth varies from an amount just appreciable by the observation of parallax (Szili, 1), to 26 diopters—8 to 9 mm.—(Lichtenstein). In Gunn's case the bottom could not be focussed with any concave lens. Usually the floor, if visible at all, is fairly clear ; in some cases it seems to be covered with a soft grey veil (Wiethe). The size varies, but is commonly between a sixth and a third of the diameter of the disc ; in one instance it occupied the whole of the inferior temporal quadrant (Bock). In this case vision was 6/24 and J. 5, not improved, in all the others vision was normal or very nearly so ; a case of Szili's, in which there was optic atrophy, apparently associated with menstrual disturbance, is not reckoned. A small paracental scotoma was noted by Stood, but was not present in the cases of Makrocki or Peltesohn, who also looked for it ; in all these cases the peripheral field was full. The condition was bilateral in one case (Stood). In no instance were other congenital abnormalities present in the same eye.

In four of the cases the walls or floor of the cavity have been pigmented. No explanation of this fact has ever been advanced, but it is a point of much importance in connection with Cases 1 and 2 of the present paper, as will be mentioned presently. In Wiethe's case the colour was black with a shade of olive green ; in Peltesohn's it was dark blue grey ; in Frost's reddish brown ; in Dyckmeester's dark with a shade of green. In some of the other cases it is uncertain whether true pigmentation was present, or only shadows from the depth of the hole (Gunn, Bock). In Thomson and Ballantyne's case there was a "gradation of shading" from the circumference to the centre. This was believed by these authors not to be a true pigmentation.*

The points of resemblance between this condition and Cases 1 and 2 of the present paper are numerous. In my cases there was a deep pit exactly at the edge of the papilla.

* Personal communication from Dr. Ballantyne.

It was filled with loose retinal elements, which would probably be almost transparent (like the retina itself) on ophthalmoscopic examination. At any rate it is unlikely that they would be sufficiently opaque to hide the pigment in the floor; they might give an appearance like the "soft grey veil" noted in Wiethe's case. The position of the pit was, on the temporal side of the nerve, and (presumably, see p. 196) in the lower part of the vertical meridian—the usual situation of "holes." Vertical sections through the middle of the papilla did not pass through it. The size and form of the nerve entrance were unaltered, and the adjacent sclera and choroid were not encroached upon. The central vessels were present in the nerve, but somewhat displaced towards the side opposite to the defect. The edges were sharp, and the cavity ran straight back into the nerve. The diameter was about one-third of the diameter of the nerve entrance. Finally, the pigment epithelium lining the walls and floor would correspond with the pigmentation observed in four of the recorded cases of "crater-like hole."

On all these grounds, therefore, I believe that Cases 1 and 2 represent that ophthalmoscopical condition. If the explanation already given of these be correct, then crater-like hole in the papilla also arises by the evagination of a portion of the secondary optic vesicle into the nerve, or, more probably, by the abnormal differentiation of a part of the neural division of the vesicle into pigment epithelium and retinal elements (see p. 214). When pigment is present it is the pigment of the retinal epithelium, when absent either the inner layer only of the secondary optic vesicle has been evaginated, or the pigment epithelium has lost its pigment, as had happened in certain areas in my cases. It has already been stated that such an abnormality has no necessary connection with the fœtal cleft. Corresponding with this, although none of the holes has been in the upper part of the disc, they have rarely been in the line of the fissure. In my cases also, although the main defect was

below, a minimal form of the same abnormality was visible on the opposite side, and a similar abnormality in this situation has been described by Monthus and Opin.

Whether certain other defects in the nerve are of the same nature is doubtful. Szili, for instance, has described three cases of sharply circumscribed pigmented defect in the centre of the physiological cup, through which the vessels entered. Thomson and Ballantyne report a case of bilateral "pigmented coloboma of the disc" in which it appeared as if a wedge had been cut out of the lower part of the papilla; it did not reach the edge, and the floor was covered with a network of brown pigment, with something of a "bone corpuscle" arrangement. Frost has figured a somewhat similar case, but with no pigment in the floor. It is possible that some of these cases are due to simple defects in the lamina cribrosa, such as were found by Görlitz. When they are pigmented, however, they are perhaps better explained as similar to Cases 1 and 2, or to a pigmented lamina cribrosa. It should be noted, however, that not all pigment spots on the disc are of this kind. A discussion of the subject will be found in a subsequent paper (p. 225).

Fuchs' Coloboma.

In a paper on coloboma at the nerve entrance some reference to the condition known as Fuchs' coloboma or inferior crescent can scarcely be avoided. As I have no personal observations to record I shall simply gather from the literature what is known as to its pathology, in order to inquire how far the lesions already mentioned are related to it.

Inferior crescent is frequently referred to as a minimal form of congenital coloboma at the nerve entrance. In support of this are adduced the misshapen, horizontally oval form of the disc itself; the abnormal course of the retinal vessels which usually enter in a downward direction; the consideration that since it occurs relatively often in hypermetropic eyes, it is improbable that it should be due to

stretching of the ocular tunics; the frequent association with astigmatism; the undoubted occurrence of border-line forms which cannot be classified with certainty as crescents or as colobomata of the choroid encroaching on the disc. The idea that inferior crescent was a minimal coloboma seemed to be confirmed by a case examined by Salzmann. Over part of the crescentic area the retina was doubled, the outer layer having a "perverse" arrangement. Salzmann regards this as evidence of a congenital origin, and as justifying its classification with the colobomata, but the fundus had never been seen with the ophthalmoscope, and in view of the conditions found in some cases of his own, Elschnig believes that it was not a typical inferior crescent. In a subsequent paper Salzmann admits that his case was not an ordinary inferior conus; he reports an ophthalmoscopically typical case in which the anatomical conditions "differed extremely little from the well known picture" of temporal crescent.

Schnabel and Elschnig both place inferior conus in a category apart from coloboma at the nerve entrance. Elschnig had an opportunity of examining three cases microscopically. He found a condition much resembling ordinary myopic temporal crescent—the scleral promontory rounded off instead of projecting in a sharp spur at the nerve entrance, the choroid, membrane of Bruch, and nuclear layers of the retina ending some distance from the nerve entrance. Above the nerve the opposite conditions obtained, the sclera and choroid projecting over the nerve entrance in the form of a sharp spur. The intervaginal space was wider on the side of the conus instead of on the opposite side, as is usual in myopic eyes, but so far as the conus itself was concerned the anatomical details were exactly similar to those found in a temporal crescent.*

* For Elschnig's account of the anatomy of temporal conus, see Arch. f. Ophth., vol. li, pt. 3, p. 391, 1900. He believes that they are due to stretching, but are sometimes founded on a congenital abnormality in the architecture of the nerve entrance. The abnormality here invoked is in the *form* of the nerve entrance; it is not a colobomá (*i.e.*, a defect), and has nothing to do with the fœtal cleft.

Elschnig believes, therefore, that inferior conus is not directly connected with the fœtal cleft, and is not strictly a form of coloboma at the nerve entrance. The essential lesion is rather a defective formation of the sclera and choroid in the lower part of the globe which allows that portion to stretch. Proof of this stretching was found in the inferior part of the globe in the form of a thinning and ectasia of the sclera exactly similar to the ectasia commonly found on the temporal side of the nerve in myopic globes. Inferior conus is the result of this stretching, just as temporal conus is the result of stretching on the temporal side of the nerve. It is possible that in some obscure and indirect manner the fœtal cleft influences the defective formation of the sclera and choroid below. This would account for the much greater frequency of conus downwards than of conus inwards or upwards. In this sense the conus might be termed a congenital abnormality, or founded upon a congenital abnormality, but not in the sense of its being a coloboma or founded upon a defective closure of the fœtal cleft. Elschnig claims that in his cases the thinness of the sclera was out of proportion to the amount of stretching, and must, therefore, have had a congenital basis. Schnabel's case seems to have been essentially the same as Elschnig's, and examples of nasal crescent examined by Elschnig and Symens were also similar.

On this hypothesis the other features of a case of inferior conus would be explained as follows. The horizontally oval form of the disc results from the pull upon it from below, due to the stretching, and corresponds with the vertically oval form so frequent in the myopic disc. The downward direction of the vessels results from the same cause and corresponds with the temporal direction of the vessels in the myopic eye. The occurrence of the abnormality in hypermetropic and emmetropic eyes is accounted for by the consideration that it is founded upon a congenital abnormality likely to be equally frequent in any type of

eye; cases of crescent in other directions in hypermetropic or emmetropic eyes may result from the occasional occurrence of a similar abnormality of the sclera and choroid in other directions, or perhaps sometimes from the stretching of a previously more hypermetropic globe. The astigmatism is a fundus astigmatism, due to the localised stretching of one portion of the posterior part of the eye. The amblyopia may be due to the impossibility of fully correcting such astigmatism, which may well be somewhat irregular; or possibly the retinal elements at the fovea are actually damaged by the stretching.

According to Elschnig, therefore, inferior conus and coloboma at the nerve entrance are really distinct entities, though it may be impossible to differentiate them in all cases with the ophthalmoscope. If the floor is much sunk below the level of the surrounding fundus, and if the defect is large and of bizarre form, these are points in favour of coloboma as against crescent.

Apart from all hypothesis, however, the fact remains that such examples of typical inferior conus as have been both seen ophthalmoscopically and submitted to pathological examination, have presented much greater resemblance to ordinary myopic crescents than to the colobomata at the nerve entrance of which examples have been given in this paper. The localised ectasia with displacement of the nerve entrance, and the snared off pockets of retina in the sclera have been absent. Nor is the difference merely one of degree. There are several examples in the literature of very small colobomata at the nerve entrance found accidentally, and these have presented lesions different from those found in inferior conus. At present, therefore, it is not justifiable to speak of inferior conus as a minimal form of coloboma at the nerve entrance. Microscopical details of typical cases are, however, still very scanty, and instances should certainly be reported as occasion arises.

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Description of Plates VI and VII, Figs. 1 to 3.

Fig. 1.—The nerve entrance in Case 1. *r*, retina; *s*, sclera; *ch*, choroid; E, colobomatous pocket cutting off the lamina cribrosa from its attachment to the sclera, and filled with distorted retinal elements; *a*, pigment epithelium in the depths of the pit; *v*, the intervaginal space; *d*, dural sheath; *l*, lamina cribrosa; *rv*, retinal vessel entering close to the upper border of the nerve; *p*, a small inclusion of pigmented tissue on the side of the nerve opposite to the main defect.

Fig. 2.—The nerve entrance in Case 2. Lettering the same. The inclusions on the opposite side of the nerve are larger, and distinctly show the structure of retina (*p*).

Fig. 3.—The nerve entrance in Case 3. *t*, temporal side of the excavation; *n*, nasal side of the excavation; *r*, retina; *r'*, retinal elements within the excavation; *v*, intervaginal space; *a*, arachnoid sheath; *o'*, spur of nerve projecting up the temporal side of the ectasia; *o*, nerve on the nasal side of the ectasia; *f*, fibrous tissue lining the floor of the pit; *p*, papilla; *l*, lamina cribrosa.

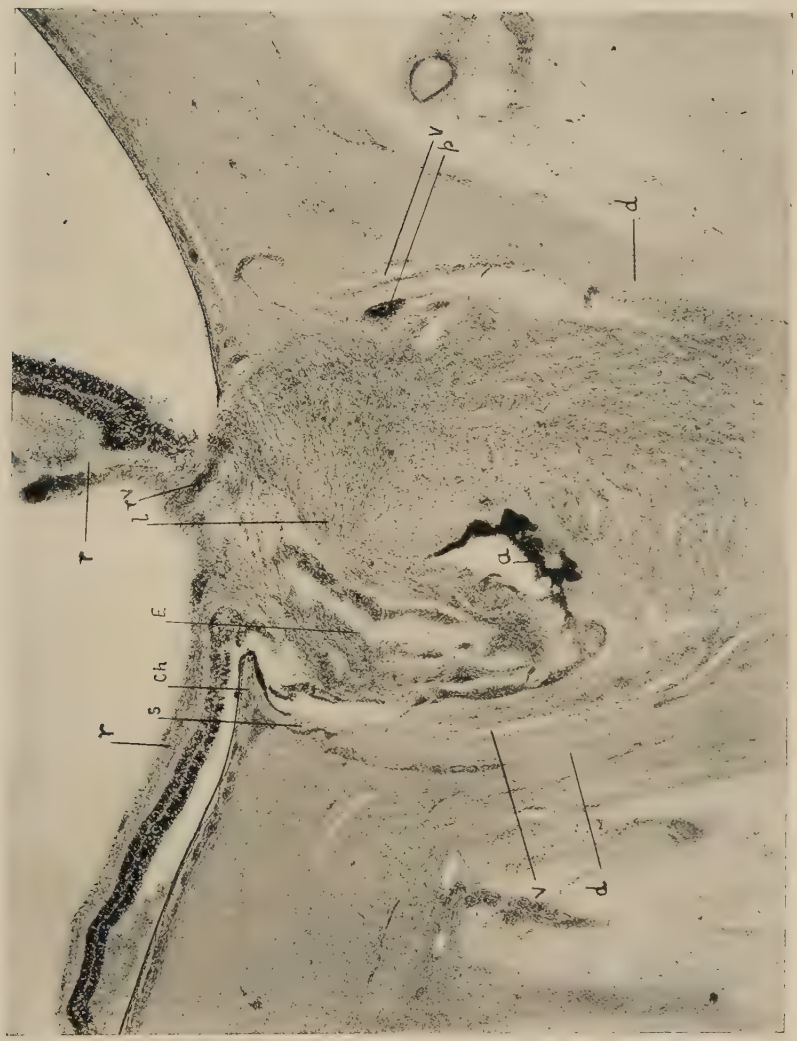

FIG. 1

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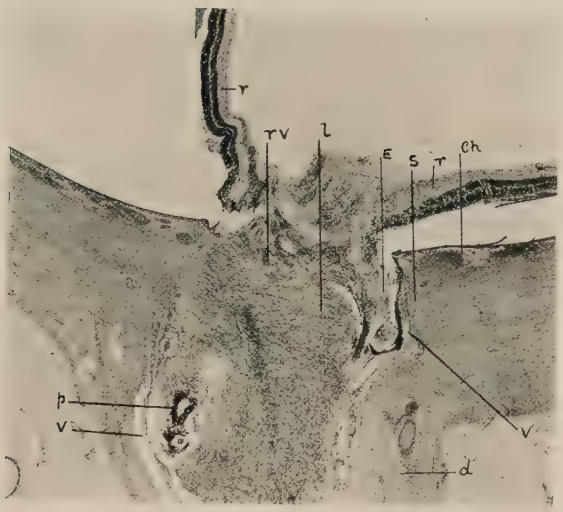

Fig. 2.

Fig. 3.

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CONGENITAL PIGMENTATION OF THE PAPILLA.

By George Coats, Curator.

(With Plate VIII.)

In connection with the subject of pigmented papilla, one form of which was mentioned in the foregoing paper, the following case seems worth recording :—

William M., aged 69 years, came to the Great Northern Central Hospital in August, 1906, on account of presbyopia. Vision :—R. $\bar{c}$. + 1 D. Cyl. Ax. H. = 6/9. L. $\bar{c}$. + 0·5 D.sph. + 0·5 D. Cyl. Ax. 40° < = 6/6 partly. With + 4 D.sph. and the Cyls. J. 1 both. The eyes were externally normal; irides blue, with a few small, flat, brownish spots. Both eyes were alike. The fields of vision were full peripherally, and no defects were found within them. The lenses showed some opacities, sufficient to account for the slight imperfection of vision. In both fundi there was evidence of moderate vascular disease, chiefly in the form of nipping of the veins where crossed by the arteries; otherwise the right fundus was normal.

In the left eye the condition shown in Plate VIII, was present. The most striking abnormality is the large pigmented patch. It is situated below, and more to the nasal than to the temporal side of the vertical median line of the disc. It occupies between a fourth and a fifth of the area of the papilla. The patch is not confined to the disc, however, but overrides it, about two-thirds being within, and one-third beyond its edge. From the superior border an elongated tongue of pigmented tissue stretches upwards immediately within the nasal margin of the disc, terminating a little above its mid-point. The nasal edge of this tongue is whitish and glittering, the temporal deeply pigmented. The area is distinctly swollen; it is difficult to focus exactly on its surface, but the amount is probably about 2 D. A large artery passes over the surface and makes a considerable bend in doing so; it stands a little away from the

pigmented mass. The main temporal vein runs over the edge of the area, and is only slightly displaced forward. Two inferior nasal veins disappear into it, and do not again become visible. The pigmented tongue on the nasal side is not raised. The colour of the swelling is pure black; there is no shade of green or brown in it. Its edges are soft and have a reddish tinge on the papilla. In an area of the fundus downwards and inwards, corresponding to the position of the pigmented area, the red colour is replaced by a soft yellow tinge. This area extends for about $1\frac{1}{2}$ disc diameters, and has very indefinite borders. It seems to be a little raised. The impression is produced that the pigmented area is larger than appears, but is partly hidden by the surrounding tissues.

The retina seems to be present over the mass, being indicated by a faint shimmering veil; the occurrence of retinal vessels in this situation points to the same conclusion. Apart from the pigmented area the disc is normally formed and of good colour; the arrangement of the retinal vessels is normal. On its temporal inferior aspect there is slight rarefication of pigment in the adjacent choroid.

Close to the disc in an upward nasal direction there is a delicate, translucent, connective tissue film, which covers, without concealing, the vessels. Its upper extremity ends in a point touching the main superior artery, the lower curves round and lies on the surface of the pigmented mass. Its upper and outer edges meet in an almost exact right angle. Its lower and inner edges form a continuous curve. No other abnormality is present in the fundus, which is of a lightly pigmented type. The degree of pigmentation is the same in both eyes.

Fourteen months after the patient's first visit there was no change in the condition.

No case in the literature seems to be in all respects similar to this. Three possible explanations suggest themselves:—(1) Sarcoma; (2) Pathological pigmentation; (3) Congenital pigmentation.

(1) Looking to the pigmented area alone, a diagnosis of early sarcoma must have been considered, on account of the swelling, the tongue of tissue sticking out from the margin, and the

altered appearance of the fundus downwards and inwards from the nerve. But in view of the presence of the other congenital abnormality—the connective tissue film over the vessels—and more especially in view of the duration of fourteen months without change, this diagnosis can be definitely ruled out.

(2) In pathological pigmentation from disease of the neighbouring nerve or choroid, a different picture is presented. Examples of this form have been examined microscopically by Alt, Wedl and Bock, and Ogawa, and in these cases, as well as in all the instances observed clinically, there was reason to suspect hæmorrhage in the nerve or its sheaths.* In consequence, pathological pigmentation usually occurs in association with optic atrophy, and vision is always very bad. In these points it contrasts strongly with the present case. Moreover, the ophthalmoscopic appearances are different. The pigment is in the form of many spots and patches in different parts of the disc, but with a tendency to be grouped chiefly in the periphery, leaving the centre free; they are not raised, and on close examination may be resolved into a collection of fine dots.

(3) It would seem, therefore, that the present case is one of congenital abnormality, a diagnosis which is strongly supported by the presence of the connective tissue web over the nasal vessels, by the normal visual acuity and full field, and by the absence of alteration during 14 months of

* The clinical evidence is as follows :—In Liebreich's case there was loss of vision, associated with sudden suppression of the menses after an accident 12 years previously. In Knapp's there was sudden loss of vision in both eyes, followed by optic atrophy, in a man aged 23 years. In Hirschberg's case the condition ensued after an injury with a piece of iron, followed by vitreous hæmorrhage. In the case of Leber and Deutschmann sight had been lost in both eyes 2 years before, while the patient was unconscious, during an attack of typhoid fever. In Ogawa's case there was pulsating exophthalmos from an injury 22 years before. The nerve ran through the middle of the dilated vessels as a thin dark cord. In Sattler's case there had been a blow on the temple, with subsequent loss of sight, 12 years before. Most of the authors mentioned attribute the condition to hæmorrhage into the intervaginal space, with subsequent diffusion of the blood pigment into the papilla, or to hæmorrhage in the substance of the papilla itself.

observation. Much more difficult is the question of its exact nature. Cases of congenital pigmentation of the papilla seem to fall into two main classes: (*a*) Isolated small black spots. (*b*) Pigmented "crater-like holes," such as have been described in my previous paper (p. 216).

(*a*) Small pigmented spots are probably not very rare, since Ogawa was able personally to observe seven cases. They are small, sharply circumscribed, usually round or only slightly elongated, and neither raised nor depressed. They occur indifferently in any part of the disc. No other congenital abnormalities are usually associated with them, and vision is perfect. In some instances the spot is in the centre of the disc, and covers the origin of the vessels (Schleich, Szili). The area may be of some extent; Liebreich's case was associated with abnormally dense pigmentation of other parts of the eye (an unusual combination), and in the centre of the disc a considerable area of "tuft-like black striæ" was present, concealing the origin of the vessels. Ogawa explains these spots as either congenitally displaced portions of the choroidal pigment, or examples of pigmented lamina cribrosa, a condition which occurs in many animals. Perhaps these two explanations are in reality the same, since in some animals (*e.g.*, the dog) the choroid takes a large share in the formation of the lamina, and the fibres which it contributes are pigmented for a considerable distance into the nerve entrance. A rudimentary degree of the same condition has been found by Ogawa in the Japanese eye, and Müller has also mentioned its occasional occurrence in the eyes of white races.

Possibly of the same nature are those cases in which the choroidal ring encroaches unduly on the disc (Jaeger, Berger, Rumschewitsch). Somewhat different, but probably allied, are instances of pigmentation of the whole disc.*

* v. Forster: Shade of dark smoked glass; eye otherwise albinotic. Hirschberg: Uniform dark slate grey, on which retinal vessels hardly visible. Hilbert: Slate grey. Pick: Dark brown, almost black, with red vessels showing on it. Opaque nerve-fibres around papilla. Mauthner states that he has seen the papilla of a sky blue, or sometimes dark brown, colour.

These cases are almost certainly due to a pigmented lamina cribrosa.

Was the present case similar to any of these forms of pigmented papilla? It differed in several important points. It was much larger than any recorded example of isolated pigment spot, and swelling has not been noted in any other instance. A more important point than these, however, is the fact that a considerable portion of it was outside the limits of the papilla. The extra papillary part amounted certainly to a third of the total area, and was probably in reality larger, since the limits of the swelling seemed to be partially concealed downwards and inwards, where the fundus was altered in appearance. For these reasons I believe that the present case was not analogous to instances of pigmented spots on the papilla.

(*b*) Was it of the same nature as the pigmented crater-like holes described in my last paper (p. 216)? The chief objection to this view is that instead of an excavation a swelling was found. It is true that some of these pigmented holes have been very shallow, and it is even conceivable that by proliferation of the pigment epithelium lining them, the cavity might become filled up and a swelling might be produced at the edge of the nerve. I do not think, however, that this explanation will suffice in the present instance. These crater-like holes, although they may cause distortion of the papilla, always lie within its limits. In my case just the opposite obtains, the papilla generally is not distorted, but the pigmented mass considerably overlaps its visible border. Moreover, in the situation of these holes the retina is distorted and partially destroyed; in my case the presence of normal retina over the mass seems to be evidenced by the shimmering veil on the surface, and by the normal course of the vessels over it. The normal vision and field do not, perhaps, count for much, since these seem to be not infrequently found in cases of crater-like hole.

The evidence seems to point to a congenital massing of

the pigment epithelium by the side of the nerve entrance, or possibly to a simple congenital tumour of the choroidal stroma pigment. If the first of these explanations be correct, the case might be compared with an enormous exaggeration of the choroidal ring. A condition in some ways analogous is occasionally found at the opposite extremity of the secondary optic vesicle—namely, the so-called melanomata of the pupillary margin. Similar structures are normally found in the horse and several other lower animals ("grape clusters," Traubenkörner).

If the tumour originated in the mesodermal pigment of the choroid it would be analogous to the pigment spots so frequently present on the iris. As is well known, these also may form raised masses, "melanomata" and "congenital nævi."* Melanomata of the choroid have also been described, having been found accidentally during pathological examinations (de Schweinitz, Purtscher ?, Fuchs ?). Such tumours consist of dense aggregations of chromatophores, and it is probable that they are benign, though there is no method of differentiating them from commencing melanotic sarcomata. Possibly this was the true diagnosis in Purtscher's example, but in de Schweinitz's case the tumour was more probably simple, while in Fuchs' there were several such patches in the same eye, a condition of affairs which almost excludes sarcoma. In none of these cases had the patch been visible with the ophthalmoscope, and in none was it found nearer than 3 mm. from the nerve entrance.

On the whole the diagnosis seems to lie between these last two conditions—an overgrowth of the pigment epithelium at the edge of the nerve entrance, and a melanoma of the choroid encroaching on the disc. Personally I should incline to favour the latter view, because of the soft edges, and the appearance of veiling of the periphery by neighbouring structures downwards and

* Some of these, however, probably originate, not from the mesoblast, but from epiblastic cells, derived from the pigment epithelium.

M. H. Brooks, pinx.

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inwards (chorio-capillaris and pigment epithelium). This seems to prove that the mass was deep in the choroid. A tumour of the pigment epithelium might be expected to end more sharply, since it would be covered by nothing but the transparent retina.

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THE VISUAL FIELDS IN RETINITIS PIGMENTOSA.

By Malcolm L. Hepburn.

The characteristic feature in the fields of retinitis pigmentosa is the presence of the ring scotoma, which has been known to exist for a long time, having been first recognised by Von Graefe in 1855, and described by him in his Archives. This defect is also frequently noticed in many other affections of the eye, but in none is it so universally met with as in pigmentary degeneration of the retina. Subsequent observers (*i.e.*, Ole Bull, de Schweinitz, Hill Griffith, Gonin, Baas, Schoen, etc.) have drawn attention to various irregularities in its shape, completeness, evanescence, etc., all of which are occasionally found in the scotoma, as well as to the fact that it is sometimes positive and sometimes relative (Hancock), or again partly one and partly the other. I have lately examined the fields of five cases of uncomplicated retinitis pigmentosa, and one other where the ophthalmoscopic picture characteristic of this condition was present over a well-defined area only of the fundus, and placed absolutely symmetrically in the two eyes. The results of these examinations are shown in the charts, and have revealed an additional variation in the ring scotoma which, as far as I can discover, does not appear to have been often noticed before (Crzellitzer, Schoen), and which may possibly throw some light on the pathology of the condition. I am well aware of the danger of attaching undue importance to the records of a purely subjective test, and on that account have accepted only those cases on whose intelligence I felt justified in relying; and I have every reason to believe that the visual fields brought forward in this paper substantially represent accurate details as seen by each patient. Possibly I might have secured some finer points in the first case, had I been able to make use of the experience gained by later examinations; I was, however, unable to induce the patient to visit the hospital again.

The fields were all taken with a McHardy's perimeter, using a 10 mm. white test object in a good natural light. I purposely avoided repeating the observations, as some other writers have done (Gonin, Hill Griffith, etc.), with feeble illumination, since in that case the points to which I wish to draw attention would not have been evident; nor did I employ different colours, as in most instances there is merely contraction of the field, and no definite purpose is served beyond the fact that colours are often clearly recognisable in cases of retinitis pigmentosa.

I am very much indebted to Mr. W. T. Holmes-Spicer, Mr. W. T. Lister, and Mr. J. H. Parsons for their kindness in allowing me to make use of and publish those cases which occurred respectively under their care.

The family history of the various cases is as follows:—

In none was there any consanguinity between the parents; the mother of Case 1 suffered from night blindness for many years before she completely lost her sight, and is now unable to see her way about, though from questions which I asked this is probably not an uncomplicated case of retinitis pigmentosa; one brother has attended Moorfields since he was 4 years old and has gradually become blind. The patient herself has had nine children, of whom three are living, but none of them has suffered from night blindness.

The mother of Case 2 has retinitis pigmentosa, and the mother's sister also cannot see well at night. One brother had to leave the Navy owing to his being unable to see at night.

Case 3 gives no family history of any importance.

Case 4 has one sister and one brother who suffer from night blindness, but neither of the parents is affected in any way.

The peculiarity in the field which I specially wish to emphasize is that in four of the cases there are small islands of vision or relatively functional areas scattered throughout the scotoma, and in addition I would call attention to (1) The fairly constant position and extent of

the central visual portion which does not seem to diminish perceptibly under ordinary illumination until the extreme periphery has entirely lost its function; (2) The few additional scotomata in the visual areas as in Cases 3, 4(R), and 6; (3) The occasional formation or tendency to formation of the double ring scotoma; and (4) The irregularities of the scotoma itself though always attacking a well-defined region in the retina.

Case 1 shows many of these islands in an otherwise very broad scotoma in the right eye, but as I have hinted above I have no doubt I might have succeeded in mapping them out with more accuracy on a second attempt, and in showing up some in the left eye. In this case there was only a small amount of pigmentation and very little change of any sort to be observed ophthalmoscopically; a point which bears out what has been frequently insisted on by previous writers, namely, that the size of the scotoma has no direct relation to the amount of pigment present (Gonin, Nettleship, Hill Griffith, Schoen, etc.).

I was fortunately able to obtain two records of Case 2 after an interval of nearly two years; and, assuming the correctness of the first observation, which I have no reason to doubt, these fields suggest one or two interesting points in the pathology of retinitis pigmentosa. The scotoma in this case is relative; that is to say, the image of the test object never completely disappeared, but was only faintly seen over the area marked out in the field. The presence of a double ring scotoma, as seen in the left eye, has been recorded twice before (Baas, Crzellitzer), and it will be noticed that at the second examination the fields appear at first sight a good deal altered; but on closer investigation the principle changes in the left eye consist of the disappearance of the extreme peripheral seeing area, the completion of the inner ring with obliteration of the small tongue passing inwards, and a visual area replacing the outer ring scotoma. The field in the right appears to have improved somewhat, the central visual area is of about the same size and shape as before, the

central triangular portion of scotoma having cleared up, and the peripheral ring of vision has materially increased. There is exceedingly little coarse pigmentation in this case, merely the fine peppery appearance of the intermediate zone with here and there some larger masses.

Case 3 is that of a young man, aged 22, who had suffered from night blindness for 7 years, and in whom the fundus showed the typical picture of retinitis pigmentosa with a very large amount of pigment in the usual situation; but no definite areas of choroidal inflammation were to be seen anywhere. The scotomata differ considerably from those of the other cases, as there is not a complete ring in either eye, though in both there is a tendency to the ring formation. In addition to the practical obliteration of the nasal part of the fields in both eyes, the right eye shows evidence of a commencing double ring scotoma, both being incomplete and the outer more so than the inner; besides which the greater part of the inner one is only relative, thus indicating a comparatively large area of functional activity in a part where one would least expect it, considering the extensive degeneration elsewhere. The left eye exhibits even greater loss of function, the only vestige of peripheral vision being down and out in the field, and the scotoma is scarcely recognisable as a ring, though undoubtedly of an annular type; it practically takes the form of a large island with a small visual area in its centre.

Case 4 was only discovered by accident in the routine examination of the eye, so that no symptoms were complained of, though on close questioning a history of night blindness was obtained. Here we have in each eye a complete ring scotoma, bearing much the same relation to the fixation point as in the other cases. The right fundus showed mostly pigment of the peppery variety, but up and in were some more typical "bone corpuscle shaped" masses, and in one part the pigment surrounded a retinal vessel; in the left eye much the same changes were visible, but the typical pigmentation was most marked down and in; and the vessels

were smaller than those in the right eye which were very little reduced in size. It will be noticed that in both eyes, but more especially the left, there are many areas of relative dimness in an otherwise complete scotoma indicating that at these spots the retina is still functionally active; there are also in this case some small scotomatous areas in the peripheral visual zone.

Case 5 is the sister of Case 4, and has suffered extreme difficulty in getting about at night, though not absolutely blind, for the last 10 years. The fundus in both eyes appeared very grey all over, the pigment was of the fine feathery variety, with typical shape and arrangement, but not very abundant, and most marked at the upper part. The retinal vessels were much reduced in size. It will be noticed that, in this case, the scotoma forms a very broad ring; and I was unable to obtain the slightest suspicion of a visual island in either eye.

I have introduced Case 6 merely to make use of as an analogy between a localised chronic choroiditis with secondary retinal degeneration over the same area, which happens to correspond with the mid-peripheral region, and cases of ordinary uncomplicated retinitis pigmentosa. This condition was also discovered accidentally and had evidently existed for some time; an examination of the fundus showed two symmetrically placed well-defined semicircular patches of old chronic choroiditis about $1\frac{1}{2}$ disc diameters broad extending from the nasal side ($2\frac{1}{2}$ disc diameters inwards from the edge of the disc) and after encircling the disc passing downwards below the macula in a curved direction towards the temporal side ending just on a level with the upper margin of the disc in an enlarged extremity, the inner border of which was situated $3\frac{1}{2}$ disc diameters from the edge of the papilla. The whole of this well-defined band is greyish in colour and covered with typical bone corpuscle-shaped pigment masses, the largest collections being concentrated at each extremity. Corresponding to this band of retino-choroiditis is a semi-circular scotoma in the field of

vision in each eye, as well as two smaller ones near the central fixation point on the nasal side in the chart of the right, and along the centre of the scotoma is a long tortuous island of vision which does not extend completely up to each extremity. An additional point of interest is that along the centre of these patches runs a retinal artery and vein.

The pathology of retinitis pigmentosa, and of the ring scotoma in particular, has always been a debatable question, and is likely to remain so, as long as the pathological material in such cases is so scanty, which can hardly be wondered at considering their nature and the rarity of securing an uncomplicated eye for examination purposes, and we are, therefore, driven to theorise from analogy and subjective symptoms. Very many opinions have been expressed with regard to the origin of the ring scotoma, and these have been well summarised and dealt with by Hancock in the Royal London Ophthalmic Hospital Reports, vol. xvi, part 4, p. 496. It is sufficient here to mention that, roughly speaking, there are two main theories, one which ascribes the defect to some affection of the optic nerve (v. Graefe, Leber, Burnett), and more especially that bundle of fibres which supplies the intermediate zone of the retina (Hancock, Lister); and the other which regards the special arrangements of the vascular supply in this region as rendering the vessels easily liable to disease or partial blocking of their lumen (Gonin, Nettleship, and others).

Gonin has explained, at some length, how the branches of the posterior ciliary arteries supplying the choroidal vessels in the mid-peripheral region are, by the absence of anastomosis with others elsewhere, left practically in the position of a terminal circulation similar to that of the fingers and toes, and as such are subject to all the weaknesses attaching to this arrangement, whereby the nutrition of the parts supplied by this system, viz., the anterior layers of the choroid and the posterior layers of the retina, may be seriously affected.

In this view Nettleship concurs, and it has, therefore, been called the Gonin-Nettleship theory. The idea of the

choroidal circulation being of a terminal nature suggested the inquiry regarding other parts of the body where the same conditions exist; and in one or two instances I was able to elicit the history of cold hands or feet. Case IV suffers from cold hands and feet, while her sister is only subject to cold hands; but Case VI complains of numbness of the ends of the fingers on the slightest fall of temperature. Moreover, it has been shown that division or ligature of the short ciliary arteries produces eventually the ophthalmoscopic appearance of retinitis pigmentosa (Wagenmann, Krückmann). One or other of these explanations has, up till now, been made to apply to all cases where ring scotoma has been found in the field of vision.

In the first place it does not seem to me at all essential to prove that all ring scotomata found in any condition are necessarily the result of a lesion in the same structure, and this thought has evidently occurred to Lister. The retina is dependent for its nourishment, like most other parts of the body, on a perfect nerve and blood distribution, either or both of which may be the seat of disease, thus seriously impairing the function of the parts which they supply.

Those who uphold the view that the defect lies in the nerve fibres regard the uniformity and regular shape of the scotoma as incompatible with the vascular theory; whereas, although the actual area of the retina liable to become involved is of an extremely uniform extent, the ring scotoma itself, which is the evidence of loss of function, is remarkably irregular both in shape, position, and completeness, points which rather support the vascular theory; and I fail to see how the presence of a double-ring scotoma can be explained on the nerve theory.

It is quite true that a lesion of any special nerve or group of fibres is followed by very definite and uniform changes in the parts supplied by it, with very little variation in the ultimate results; whereas if a vessel in other parts of the body happens to become cut off from any cause, the neigh-

bouring circulation immediately attempts to compensate for the break in continuity, and the resulting changes are different in character and vary in severity.

Another point urged against the vascular theory is that the special arrangement of the blood vessels, which is supposed to render them susceptible to disease, does not exist in that part of the retina corresponding to the position of the scotoma, but at the equator, which is 67° outwards from the fixation point (Hancock). Provided Hallidie's calculation is correct, this is an argument in favour of the nerve theory; but it does not follow that, because the vortex veins pierce the sclerotic at the equator, therefore the terminal capillaries of the posterior ciliary vessels are necessarily opposite the same area.

There are, however, some clinical cases which certainly support the nerve origin of the affection; for example, Hancock's first case, Burnett's case alluded to by Hancock, though by no means an uncomplicated one, and possibly also those of glaucoma. On the other hand, there are undoubtedly cases met with clinically which are best explained on the theory of vascular obstruction.

The pathological defect in syphilitic disease generally is acknowledged to be one which affects the blood vessels, and when properly treated the lesions which are thus caused can be made to recover completely, and the normal circulation is restored. Cases of specific retinitis, choroiditis, and retino-choroiditis, cited by Ole Bull and others, show evidence of temporary disturbance of function by the appearance of various scotomata in the field of vision which may also assume the annular shape. By suitable treatment these are dispersed, and the retina at these spots becomes functional again; this can only occur by recovery of the circulatory apparatus.

In more chronic forms of retino-choroiditis and retinal degeneration similar scotomata are seen, and the irregularities they show are much more consistent with the vascular theory, seeing that the fault lies in a tissue where

at one time there may be some vessels affected while others escape.

The islands of vision which I have noticed in the ring scotoma of cases of retinitis pigmentosa give additional support to this view.

This dual explanation of the cause of ring scotoma will, I think, account for the varying results which have been obtained by different observers from microscopical examination (Lister, Nettleship, Leber, Aubineau, etc.).

With regard to these islands of vision scattered throughout the annular scotoma there is one other point worthy of consideration. Do they represent portions of functional activity remaining in an otherwise completely blind area, indicating that some few vessels have escaped disease; or are we to regard them as definite parts of an originally complete ring scotoma which have once more become functional owing to a new blood supply from another source, *e.g.*, the retinal vessels?

It is fairly definitely proved that physiologically there is no anastomosis between the retinal and choroidal circulations except in the neighbourhood of the optic disc, and that only of an entirely capillary nature (Parsons, vol. iii); also that the rods and cones are supplied by the choroidal circulation. But I am inclined to think that Cases 2 and 6 present some strong evidence in favour of the suggestion of a new vascular supply. The presence of the retinal artery and vein along the centre of the choroidal patch in Case 6 corresponds to the line of visual area running along the middle of the scotoma, so that it seems possible that the relationship between the two circulations is so close that on an emergency the retinal may take the place of the choroidal, when, owing to disease, the latter is cut off from supplying the rods and cones. It is more than probable that this may also account for the fact that some cases of retinitis pigmentosa progress so slowly, depending on the amount of compensatory enlargement which the retinal vessels undergo; and, moreover, that the ultimate prognosis may be largely

influenced by the condition of these vessels, which constitute another example of a terminal vascular system.

Conclusions.

1. In most of the cases of retinitis pigmentosa which I have examined there are found islands of vision, or relatively functional areas, scattered throughout the ring scotoma.

2. The manifestation of retinal activity in this particular part of the field is more in conformity with the vascular theory brought forward to explain the existence of the ring scotoma (Gonin, Nettleship).

3. There are some grounds for believing that, in an emergency, when the circulation from one system (viz., short ciliary vessels) is cut off, a part of the retinal circulation is capable of enlarging in order to take its place and preserve the function of some parts of the retina.

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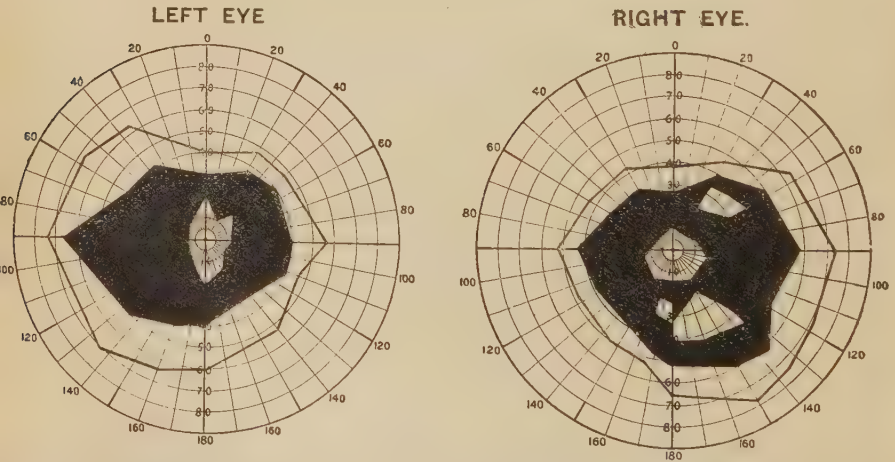
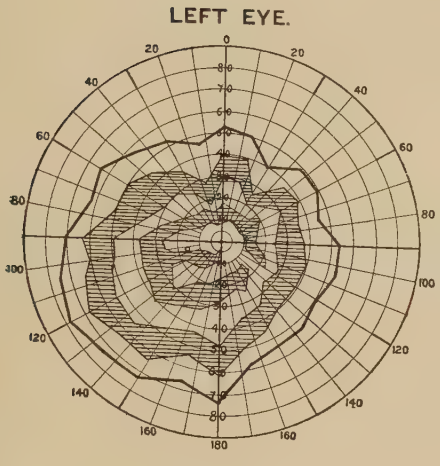
Description of the Cases.

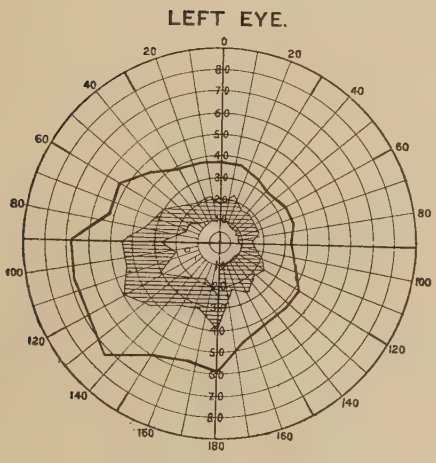
Lightly shaded areas = relative scotoma; darkly shaded areas = absolute scotoma.



Case 1.—Emily B., aged 45, under the care of Mr. Lister at the London Hospital. Retinal vessels somewhat reduced in size.

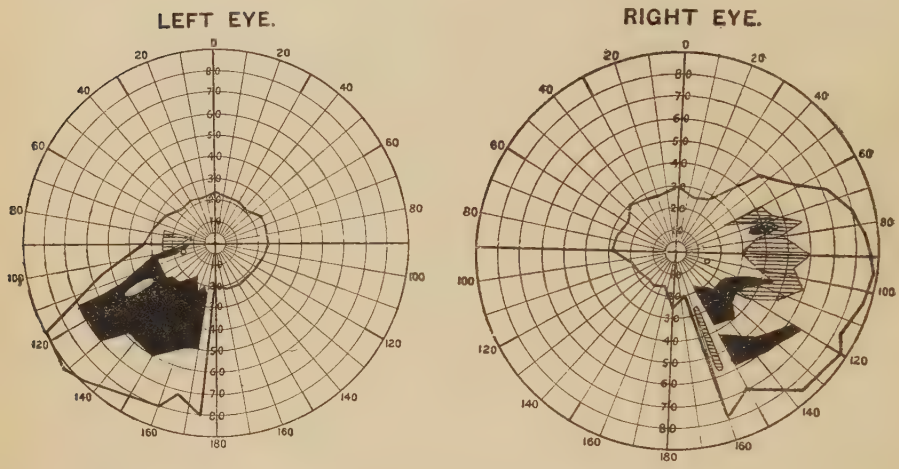


Case 2 (*a*).

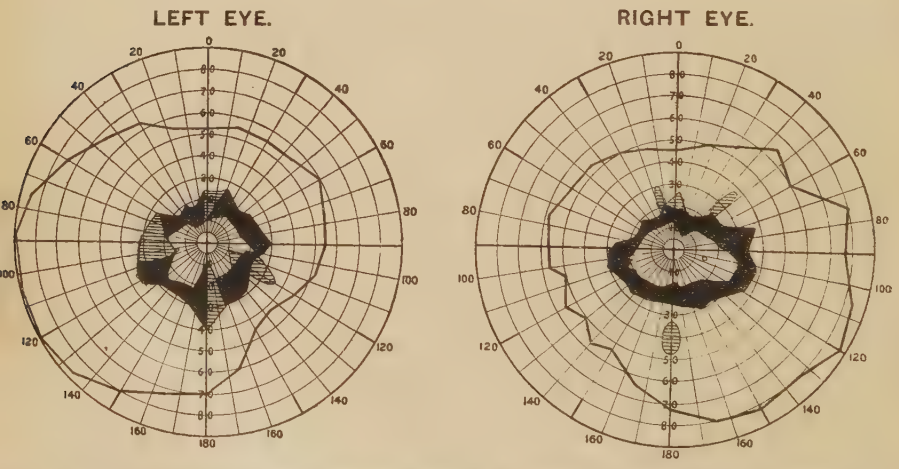


Case 2 (*b*).

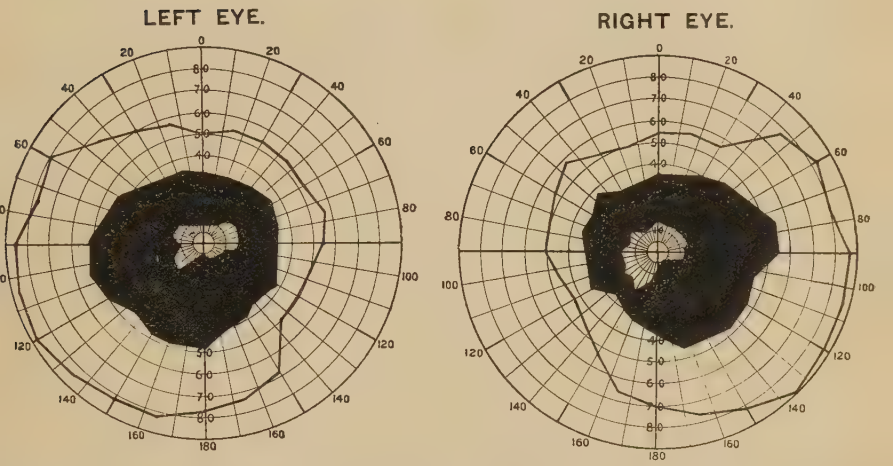
Case 2.—Wm. B., aged 19, under the care of Mr. Lister at the London Hospital. Sympt have been noticed since he had typhoid fever, six years ago. Pupils equal and act, but not well maintain contraction. R.V. 6/6. L.V. 6/12 pt. Retinal vessels *not* diminished size. (*b*) was taken two years after (*a*).



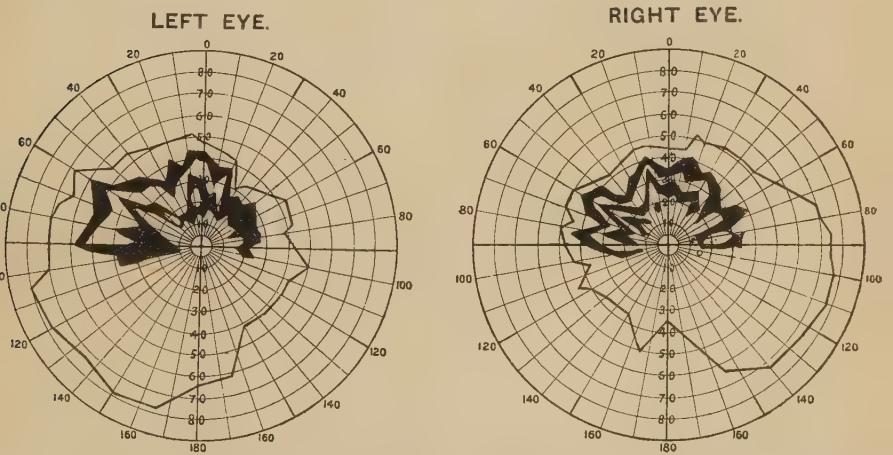
Case 3.—George H., aged 22, under Mr. Holmes Spicer's care at St. Bartholomew's Hospital. R.V. and L.V. 6/6. No H.M. Pupils equal and act well to light, and contraction fairly well maintained. The retinal vessels are quite the normal size.



Case 4.—Lily S., aged 25, under the care of Mr. J. H. Parsons at the Roy. Lond. Ophth. Hosp. R.V. and L.V. 6/6, with correction, which is + ·75 cyl., axis horizontal. Pupils equal, but do not act well to light nor maintain their contraction. Vessels only slightly diminished in size.



ASE 5.—Jane S., sister of Case 4, aged 36. R.V. 6/36, with +2·5 sph. = 6/12, and the same with the left eye. Right pupil acts better than left, and both maintain their contraction fairly well. Retinal vessels much reduced in size.



ASE 6.—George P., aged 64, under the care of Mr. Holmes Spicer at St. Bartholomew's Hospital. R.V. 6/60, with +3 sph. = 6/9; the same in the left eye. Six years ago he had facial paralysis, which still remains; there is no specific history. Retinal vessels quite the normal size.

LACHRYMAL ABSCESS IN THE NEW BORN.

(Being part of the Jacksonian Prize Essay, R.C.S., 1904.)

By M. S. Mayou.

(With Plates 9 and 10.)

Lachrymal abscess occurs during the first few weeks of life, and, as in adults, is primarily due to obstruction of the lachrymal duct with a secondary infection from ophthalmia neonatorum or organisms which may gain entrance to the lachrymal sac. In a paper in the Ophthalmic Review, 1900, Donald Gunn described several of these cases.

Recently I have had the opportunity of treating eight cases of this disease, and in connection with them have worked out some points in the development of the lachrymal canals in the fœtus, with their condition at birth, and have made some experiments with regard to the production and position of the abscess.

Development.

The fœtuses were hardened in formol, imbedded in celloidin or paraffin, and cut in serial section; where decalcification was necessary, sulphurous acid (B.P.) was used.

The following are the lengths and ages of the fœtuses examined :—

Length from occiput to buttock.	Approximate age, ascertained from history, measurement, and relative development.
4½ c.m.	10th week.
7 „	3rd month.
9 „	4th „
11 „	4th „
20 „	5th „
20 „	5th „
21½ „	5th „
24 „	5th „

And five fœtuses at full term in a good state of preservation.

OPHTHALMIC

Where injections of the duct were made, they were performed on the fresh fœtus before hardening.

The canaliculi.—The canaliculi are formed shortly before the tenth week, probably by an infolding of the epithelium of the lid margin, which is thrown into numerous horizontal furrows shortly before union takes place. At the tenth week the canaliculi consist of solid columns of cells, opening into the lachrymal sac. The canaliculus at this time is about twice the diameter of the lachrymal duct towards its lower end, and, indeed, it remains relatively large until shortly before birth, at which time it is much about the same size.

The character of the epithelial cells lining the canaliculi can easily be distinguished throughout fœtal life, as they are much larger, cuboidal in shape, more approaching the squamous type, than those lining the duct, a difference which becomes more marked as the fœtus grows older.

At the twelfth week the canaliculi have developed a well-marked lumen, and are lined by three or four layers of squamous epithelium. This lumen, from that time forward, gradually diminishes in size from epithelial proliferation, although the size of the canaliculus increases. At birth, the lumen is almost occluded by epithelial cells of a squamous type, many of which are in a state of degeneration and desquamation. The fœtal thus differs from the adult canaliculus in being lined by epithelium of two or three cells in thickness and more or less columnar in form.

The lachrymal sac and duct are developed in the course of the facial cleft as an infolding of epiblast; they occupy only a small portion of the cleft. The position of the duct up to about the sixth month is much farther forward than the eye, owing to the lateral position of the eyes in the head of the fœtus, thus resembling the condition found in the lower animals. The lower end of the duct is the first part to be differentiated, but it does not acquire a lumen until the fourth month of fœtal life. Bone is first deposited around it at the lower end, commencing internally and gradually surrounding it. Unlike the lower end of the duct,

the lachrymal sac practically never loses its lumen; it is the last part of the facial cleft to close. The lumen is of large size, gradually tapering down to the lower end of the duct, which, as has been shown, is very small.

As the bone is gradually formed around it, appearing first on its inner surface, the duct by no means occupies the whole of the bony canal, and even at the birth there is a certain amount of connective tissue around it. The epithelium lining the duct is of the same character as that lining the nose, being of a columnar type.

Anatomy at Birth.

This was studied by sections through three fœtal heads in a horizontal direction. The fœtuses examined were hardened by the injection of formalin, frozen in ice and salt, and cut with a fine saw. Some of the ducts were first injected with glycerine jelly coloured by washing-blue, and with melted paraffin coloured with sudan.

At birth only a small portion of the inferior meatus of the nose, close to the septum, is patent. The turbinated bones are packed close together, the inferior resting on the floor formed by the palatal process of the superior maxilla and palate bones (Plate X, Fig. 3). The interstices of the turbinated bones are filled with *débris* from the desquamating epithelium. In a similar manner the lachrymal duct and the canaliculi are filled with *débris*.

The lower end of the duct is about the same diameter as the canaliculus, and will barely admit a fine bristle. It opens behind the inferior turbinated bone, on the outer wall of the nose. The opening is slit-like and arranged antero-posteriorly, with a free margin to the inner side, forming a sort of valve; it is covered in by a triangular piece of the turbinated bone, which is pressed up against it laterally by the floor of the nose, on which the bone rests.

Passing upwards the duct rapidly enlarges, and when distended with paraffin measures 1·75 mm. in diameter, about the middle of its course. It joins the sac, which is

very little larger than the lumen of the duct, by a very tapering neck, so tapering in fact that it can hardly be distinguished at all. The sac itself tapers off above, ending about 1 mm. above the internal palpebral ligament. At the upper end the sac is not very near the bone, there being a considerable space behind.

Lower down at the entrance of the bony canal the sac lies close to the bone. The lachrymal bone in this region is thin, a point to be remembered when passing a probe. If a probe were pushed through this wall it would open into the infundibulum. Lower down the duct becomes imbedded in solid bone, with a considerable interval between it and the nasal cavity, until near the end of its course, where it opens obliquely into the inferior meatus, beside the triangular portion of the inferior turbinated bone, which is pressed up against it.

The nasal duct at birth is very short, not measuring more than 7 mm. (bone canal) (Plate X, Fig. 3). A probe passed through the lower canaliculus, and resting on the floor of the nose = 2 cm. Its direction is much more backwards than it is in the adult, a probe making an angle of about 45° with the forehead when passed through the duct. The shortness of the duct and its backward direction is probably due to the non-development of the air sinuses, which at birth are only rudimentary.

To test the patency of the duct at birth, injections were made into four lachrymal ducts, by first dilating the lower canaliculus with a Nettleship's probe and then syringing through with warm jelly, such as is used for mounting macroscopic eye specimens, coloured with washing-blue. The syringe was passed with every care until the duct was reached, and then the injection was made. In two instances the fluid did not reach the nose at all, extravasation taking place; in the other two the fluid only reached the nose after prolonged gentle syringing. Whenever an at all forcible injection was made it always resulted in extravasation. Sections, both microscopic and macro-

scopic were made of these (Plate X, Fig. 4). The extravasation invariably took place in the same region as that in which a lachrymal abscess is formed.

In the upper part of the groove in which the lachrymal duct lies, above the internal palpebral ligament, the injection passed directly around the sac. In the lower part, between the lower internal palpebral ligament and the entrance of the duct into the bony canal, the extravasation took place forwards into the lower lid. The reason for this is obvious on examining the sections.

The fascia covering the internal rectus muscle passing forwards gives off a slip which blends with the periosteum of the posterior lip of the lachrymal groove, whilst it passes forward as the internal check ligament, to be attached to the eyelids and orbital margin below, whilst next the muscle it blends with the capsule of Tenon (Plate X, Fig. 4).

This fascia, therefore, bounds a triangular space with its apex directed backwards, the inner wall being made up of the periosteum of the lachrymal bone, and the attachment to it of the slip of the internal check ligament, the outer wall being formed by the internal check ligament, whilst the anterior wall or base of the triangle is formed by the skin and orbicularis muscle, etc., and is crossed by the internal palpebral ligament.

The lachrymal sac is situated on the inner wall of this space, supported on its inner and posterior surfaces by bone, so that when infection takes place an abscess forms in the space described, the result being that the pus, unable to pass backwards into the orbit on account of the attachment of the internal check ligament to the lachrymal bone, makes its way forwards, generally below, but occasionally above the internal palpebral ligament, and points through the skin. Directly behind the attachment of the internal check ligament is the orbital fat; the great importance of the integrity of this internal check ligament as preventing orbital cellulitis, is thus seen.

Conclusions.

It is thus seen:—

1. That throughout development, the lower end of the lachrymal duct is extremely small, and remains so at birth, being partially occluded by the pressure of the inferior turbinated bone.

2. That the duct at birth is filled with *débris* both from the sac and the canaliculi.

3. That at birth the duct is either not patent at all, or only partially so.

After the birth of the child the nose soon becomes cleared of *débris*, probably by aspiration, but in the case of the lachrymal duct the process probably takes much longer, especially as the lower end is so small and is partially occluded by the inferior turbinate. In cases of this variety of congenital lachrymal obstruction the aspiration from the nose probably fails to clear the duct.

This is further borne out by the clinical fact that, if one of these cases of lachrymal obstruction in infants be seen before an abscess forms, one careful passage of a probe is generally sufficient to effect a cure.

It has also been suggested that some of these cases are due to the persistence of a normal cartilage of fœtal life in the nose of the child at birth, but I could find no evidence of any such cartilage. In severe deformity of the facial cleft, lachrymal obstruction does occur, but apart from such deformity I think that it is generally due to the condition described.

If infection of the sac wall takes place in one of these cases of lachrymal obstruction, an abscess forms. This frequently takes place in these cases owing to the liability of infants at birth to infection of the conjunctival sac, the *débris* making an excellent medium for the growth of organisms.

The peculiarity of a mucocele in the newly born is the large size which it reaches without showing much external

sign (Plate IX, Fig. 1). This was attributed by Gunn to the dilatation of the duct. Against this it has been shown that there is a large space behind the upper end of the sac, the duct occupying only a part of the cavity. The infant's cheeks are usually fat, and therefore liable to mask swelling. In three cases, in which I have carefully stuffed the cavity with iodoform gauze, and then had it X-rayed, there was no dilatation of the duct seen, the gauze appearing in front of the maxillary bone; further the bony wall of the duct is firmly ossified at birth, and it is hardly conceivable that it could expand (Plate IX, Fig. 2). In two of the cases which were examined there was no evidence of any swelling in the nose.

The large mucocele, with regurgitation into the conjunctival sac, renders these cases liable to be mistaken, on superficial examination, for ophthalmia neonatorum, but their true nature is readily revealed by pressure over the lachrymal sac.

Bacteriology.—The following organisms were found in the purulent discharge from these cases:—(1) Gonococcus: (2) Staphylococcus; (3) Pneumococcus; and (4) Morax Axenfeld bacillus. In all the cases the cultures were not pure, more than one organism being present.

The treatment adopted was to cleanse the sac and pass a probe.

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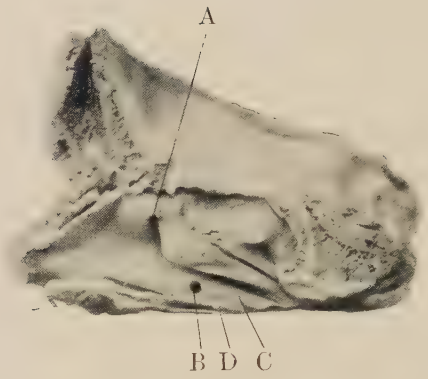

Fig. 3

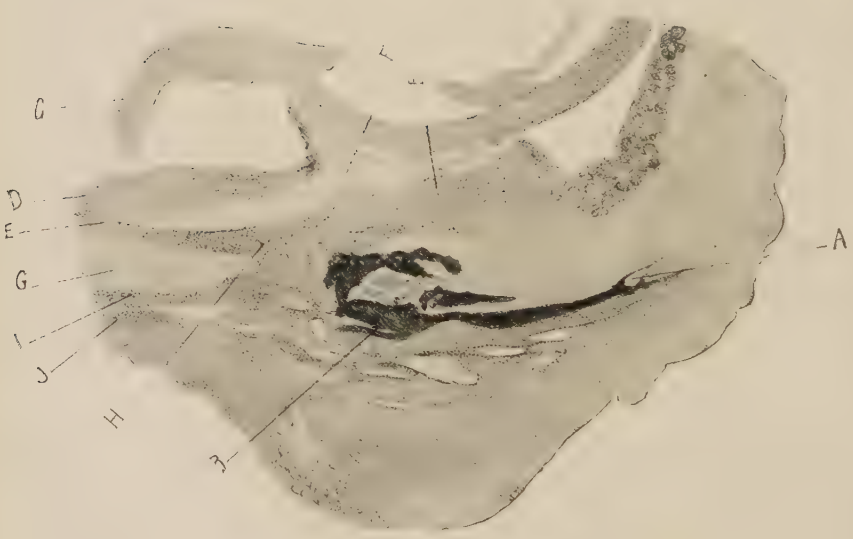

Fig. 4.

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Description of Plates IX and X. Figs. 1 to 4.

Fig. 1.—A case of lachrymal abscess in the new born, showing the comparatively slight swelling in the lachrymal region.

Fig. 2.—The abscess cavity of the same case, stuffed with iodoform gauze and radiographed, showing the cavity entirely in front of the maxillary bone.

Fig. 3.—The outer wall of the nose in the new-born child.

A is placed over the situation of the upper end of the duct.

B over the lower end.

C. The inferior turbinate bone, which presses both on the floor of the nose and lower end of the lachrymal duct.

D. The floor of the nasal cavity.

Fig. 4.—Horizontal section through the lachrymal region of a new-born child at the extreme upper limit of the lachrymal sac, which has been injected with coloured jelly.

A. The eyelid.

B. Extravasated coloured jelly around the lachrymal sac.

C. The sclera.

D. Internal rectus muscle.

E. The fascia covering the internal rectus muscle attached to the wall of the lachrymal groove H.

F. Tenon's capsule.

G. Orbital fat.

I. Periosteum.

J. Bone of the inner wall of the orbit.

SYMPATHETIC OPHTHALMIA: SOME ANATOMICAL CONSIDERATIONS, WITH SPECIAL REFERENCE TO THE OCCURRENCE OF PLASMA CELLS.

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The following notes were made as part of some researches into the histological changes that take place in sympathetic inflammation. Since the publication of Professor Fuchs' paper ("Über sympathisierende Entzündung, nebst Bemerkungen über seröse traumatische Iritis," Archiv für Ophthal., 1905, vol. lxi, part 2, p. 365), very little has been added to our anatomical knowledge of this subject, and I shall therefore quote his results as a basis for my investigation. I shall make a summary of his paper, in so far as the histology is concerned, leaving out the part where he deals with statistics.

The method of investigation adopted by Professor Fuchs was the following:—Out of his very large collection of pathological material, he first made selection of all those specimens which showed changes that he considered characteristic of sympathetic inflammation. He then referred to the clinical history of each case, and found that in all of these (with one exception, in which *irritation* had been present) sympathetic inflammation had actually existed.

In this way he found himself supplied with 35 cases, in which not only were the typical anatomical conditions present, but also sympathetic inflammation had existed in the other eye. An agreement between the anatomical and clinical conditions, confirmed without prejudice in this way, proved beyond doubt that, in sympathetic inflammation, characteristic anatomical features are to be met with.

With regard to the clinical history of these 35 cases—in 24 there had been accidental trauma, in 2 a perforating ulcer, in 4 an extraction for cataract (in 3 of which there

had been escape of vitreous at the time of operation), in 1 an iridectomy for glaucoma, in 1 an operation for anterior staphyloma, and in 3 sarcoma of the choroid with necrosis.

The features which may be looked upon as typical of sympathetic inflammation are considered under two headings, viz.:—

I. Cell Forms.
II. Their Distribution in the Tissues of the Eyeball.

I. The Cell Forms which constitute the characteristic infiltration are the following:—

(i) *Small Mononuclear Round Cells*, i.e., *Lymphocytes.*—These are invariably present. They correspond to the small round-cell infiltration found in all chronic inflammation of tissues; and they resemble, if they are not actually identical with, the lymphocytes of normal blood.

The lymphocyte has little or no protoplasmic body. A slight band of protoplasm at each side of the nucleus may sometimes give rise to the description "signet-ring nucleus." The nucleus stains very deeply with hæmatoxylin. When stained with polychrome methylene blue, the chromatin is seen to be collected into distinct clusters.

The cells occur isolated, or in nodes which are easily recognised under a low power, and constitute the so-called "patchy infiltration." They appear first in the region of the vessels, either singly or as minute nodules. These nodules are most easily observed in the walls of the veins, and are seen either in the straight portion or at the fork of a branching vessel. They gradually increase in size and completely surround the vessel. Adjoining nodules coalesce and the infiltration becomes more "diffuse."

(ii) *Epithelioid Cells.*—These are a very constant feature. The epithelioid cell possesses an extensive protoplasmic body, and a large, pale, oval nucleus, which may be excentric in position. There is almost invariably a distinct nucleolus, and pigment granules are frequently met with in the cell. The cell-wall is sometimes sharply defined in its outline, but

more often it is indistinct, when the protoplasm seems to dissolve away at the edges. Neighbouring cells in this way may often seem to coalesce (without becoming a giant cell).

The origin of epithelioid cells is multiple.

(*a*) *From the Normal Cells of the Uveal Stroma.*—The metamorphosis can be traced when the stroma cells are pigmented. The chromatophores gradually become plumper in outline, lose their processes and their pigment, and finally assume the epithelioid form.

But in the case of the unpigmented stroma cells there is considerable difficulty in following the development. When, however, a group of unpigmented epithelioid cells are met with in the midst of a mass of lymphocytes, the surrounding pigmented stroma cells being unaltered, the probability is that they have arisen from the unpigmented stroma cells (*e.g.*, in the choroid from the endothelioid cells which lie upon the elastic network).

(*b*) *From the Endothelial Lining of the Vessels.*—The epithelioid cells of the infiltration are frequently seen to possess the concentric arrangement which is met with in the cells constituting the vessel wall.

(*c*) *From the Retinal Pigment Epithelium of the Uvea.*—The lymphocytes of the infiltration become very numerous in the stroma of the uvea, and, gradually forcing themselves against the epithelium, insinuate themselves between the individual cells of the latter. In this way the epithelial cells become loosened and detached. They gradually lose their original cylindrical or cubical shape and become epithelioid in shape. The original pigment becomes set free in the surrounding media, and the cells in the mature stage are more or less devoid of pigment. On the other hand, however, the cells, instead of losing their pigment, may acquire an additional amount to such an extent that they appear quite black. Professor Fuchs thinks that this new pigment is formed endogenously, and is not merely ingested from without. The proof for this he finds in the fact that these pigment-ladened epithelioid cells are found at times in

regions where not only are there no loose granules in the surrounding tissue, but no normal pigmented cells in the vicinity (*e.g.*, in one case of scleral rupture, typical sympathetic infiltration was found in the cicatrix and in the episclera. In the latter situation many of the epithelioid cells were pigmented. These cells could only very remotely have originated from the pigmented cells of the uvea; and, therefore, their pigment was probably due to an inherited power of manufacturing pigment within the cell).

The invasion of the epithelium by the lymphocytes is observed more frequently in the iris than in the ciliary body and choroid, since in the two latter the resistance offered by the lamina vitrea is very considerable. Epithelioid cells arise at a stage later than the first invasion by lymphocytes. The earliest and smallest foci of sympathetic infiltration, as a rule, consist of lymphocytes alone; it is only in the larger later ones that the epithelioid cells are manifest. The groups of these cells may become so large that the lymphocytes are forced to the edge of the nodule; and, in preparations stained with hæmatoxylin and eosin, one therefore sees the reddish islands of epithelioid cells surrounded by deep blue lymphocytal areas.

(iii) *Giant Cells.*—These are a frequent, but not an essential feature. (Professor Fuchs met with giant cells only in half his cases.) They are derived from epithelioid cells—either by growth of a single cell, with multiplication of the nuclei, or by fusion of several adjoining cells. The nuclei take up different positions in the cell, central, crescentic and ring-shaped grouping being the most usual. Pigment is generally, although not always, present. Giant cells may occur singly or in groups. In some cases they occur in extremely large numbers, in others they are entirely absent. When present, they are found, as a rule, at the parts where the epithelioid cells are most abundant.

(iv) *Mast Cells.*—Occur in many cases, but sparingly.

(v) *Polynuclear Leucocytes.*—These are rarely met with in characteristic infiltration. When they occur they may

be looked upon as the expression of an accompanying non-sympathetic inflammation.

(vi) *Russell's Corpuscles*, i.e., *Hyaline Globules.*—These are very frequent. They are found in inflammatory conditions of the tissues everywhere.

(vii) (?) *Plasma Cells.*—Professor Fuchs speaks of certain cells which occur at the thinned-out margin of the larger lymphocytal nodules. They have abundant protoplasm which is *often stained deep red with eosin.* When stained with methylene blue, the nucleus shows large, well-marked, chromatin granules "of such a sort as to allow the cells to be classified with the plasma cells of Unna."* No further mention is made of these cells.

II. The Distribution of the Cellular Infiltration.—The region essentially involved in sympathetic inflammation is the uvea, *i.e.*, iris, ciliary body, and choroid.

(i) *The Iris.*—Although clinically the symptoms are most pronounced in the iris, yet histologically the infiltration is found to be insignificant. Out of Professor Fuchs' 35 cases the iris in four was scarcely at all infiltrated; in 11 it was affected equally with the rest of the uvea; in three† it was affected more than any other part. In no case was the iris the only part involved. The infiltration begins in the posterior segment, even behind the level of the sphincter. It consists at first of lymphocytes, but epithelioid cells appear later. The nodes are isolated at first, but later coalesce, and form a more or less continuous layer in the posterior segment of the iris. The infiltration tends to pass backwards rather than forwards, so that the

* *Loc. cit.*, p. 380.

† These were cases in which the infection had entered *viâ* the iris itself: *e.g.*, one case of ulcus serpens with prolapse of iris, the prolapsed portion showing the greatest infiltration; another case was that of sub-conjunctival dislocation of the lens, with prolapse of the iris under the lens; the third case was an extraction for cataract, in which the iris had healed into the wound. But this is not necessarily the case, for often one finds the iris practically exempt from infiltration, although the original path of infection lay there (*e.g.*, iridectomy).

anterior segments, even in advanced cases, may be exempt. Occasionally, however, in severe cases, the whole stroma of the iris may be diffusely infiltrated. The lymphocytes and epithelioid cells as they increase in numbers force themselves more and more against the epithelium of the pars retinalis iridis. They insert themselves between the epithelial cells, so that the latter eventually become disorganised.

The posterior pigmented layer is somewhat more resistant than the anterior, and, therefore, often stretches out for some time behind the mass of infiltration. This mass has thus come to occupy the space between the two layers of retinal epithelium, *i.e.*, the space of the secondary optic vesicle. Subsequently, however, the posterior layer gives way, and the infiltration is no longer confined to the iris, but extends into the posterior chamber of the eye.

The various stages in this process of extension backwards are well mapped out by hæmatoxylin and eosin. Under a low power one distinguishes the early nodules of lymphocytes as isolated dark blue spots in the posterior segment. Later these become larger, or may coalesce and form a broad band. The advent of epithelioid cells is indicated by the presence of little reddish areas in the blue nodules, or later as a continuous reddish band in the most posterior segment of the infiltration.

Giant cells occur in the iris, but are less frequent than in the ciliary body or choroid. At first they are confined to the posterior layers, but later may be met with throughout the stroma, in consequence of the wider distribution of the epithelioid cells.

The blood-vessels of the iris resist the action of the infiltrating cells for a long time (being chiefly thick-walled arteries), and thus stand out to view as bright refractive rings in the midst of the infiltration. Ultimately, however, they become more or less obliterated.

(ii) *The Ciliary Body.*—This is affected in every case, but sometimes only to a slight extent. (Professor Fuchs

believes cases might occur in which it was exempt.) The course of the infiltration process is best seen in the posterior part of the pars plicata. The epithelium of the pars ciliaris retinalis rests upon the vitreous membrane. On the outer side of the latter there is a broadish band of somewhat condensed connective tissue supporting the veins of the ciliary body. This is known as the "inner zone" of the pars plicata. The tissue lying to the outer side of this zone is much less condensed, and reaches up to the inner surface of the ciliary muscle, sending processes also among the muscular fibres and bundles. This is the "outer zone" of the pars plicata.* The infiltration begins in the outer zone, and is at first lymphocytal. The inner zone then becomes infiltrated, but chiefly with epithelioid cells. Epithelioid cells are formed in the outer zone also, but are not easily discerned in the dense masses of lymphocytes. The epithelioid cells become rapidly numerous everywhere, and sometimes out-balance the lymphocytes. In hæmatoxylin and eosin preparations one sees at an early stage the broad blue band of lymphocytes in the outer zone. Later reddish areas appear in the inner zone, and the blue band becomes broken up by reddish islands. Lymphocytes also pass in from the outer zone, and may be seen as blue strands which break up the continuity of the epithelioid infiltration. The cells then attack the retinal epithelium. This is protected, however, by the elastic tissue of the lamina vitrea, and the invading cells have more difficulty in reaching the epithelium than was the case in the analogous layer of the iris. After a time, however, resistance is overcome, and the lymphocytes and epithelioid cells may be seen on the inner aspect of the membrane (with or without visible break in its continuity). These cells accumulate and invade the posterior pigmented layer of the epithelium in the usual way. The inner or unpigmented layer, however, resists still longer, and may be seen as a definitely recognisable epithelial

* Normally, the boundary between the two zones is not very definite, and it must not be expected to be any more so in case of inflammation.

layer on the surface of the infiltration. The posterior epithelium having become demolished, the infiltration now occupies the space which corresponds to the secondary optic vesicle—as in the iris. Finally the unpigmented epithelium itself may become destroyed, and the infiltration passes out into the vitreous, forming the cyclitic membrane. The muscle of the ciliary body is often unaffected, but occasionally the lymphocytal invasion may extend outwards into it. The muscle may become destroyed in this way, but as a rule, even in severe cases, it is fairly intact.

The vessels of the ciliary body being chiefly venous and thin-walled, disappear early in the process of infiltration.

Giant cells occur chiefly in the inner zone, since it is here that the epithelioid cells are most abundant. They may lie singly or in rows along the inner part of the inner zone.

It is evident that there is a well-marked correspondence between the iris and ciliary body in this process. In both regions the infiltrates appear first in the segments that look towards the centre of the eye. These infiltrates tend to pass *inwards*, and the outer segments may remain exempt. It is in the inner (*i.e.*, in iris, the posterior) segments of the infiltration that epithelioid cells are most abundant, and in the outer, lymphocytes. In both, the lymphocytes and epithelioid cells tend to pass through the outer layer of the retinal epithelium before the inner, and having disorganised the former, become heaped up in the space that corresponds to the secondary optic vesicle. The destruction of the remaining layer is, in the ciliary body especially, a matter of considerable difficulty.

(iii) *The Choroid.*—It is here that the characteristic features are most marked. Out of his 35 cases, Professor Fuchs found that in 22 the choroid was intensely affected.* In the remaining 13 cases the choroid was affected less than

* In 12 of these 22 cases the choroid was affected more than the rest of the uvea (the latter, in three, being scarcely at all involved). In the other 10 cases the choroid was affected at least as much as the rest of the uvea.

the rest of the uvea (indeed, in three it was practically exempt). Professor Fuchs has never met with a case in which the choroid was entirely exempt. The extreme case of affection of the choroid would be "sympathetic choroiditis"—examples of which are known to occur. The infiltration is at first, as usual, patchy. It is more marked in the posterior segment than in the anterior. The region just behind the ora serrata is the least affected, and may be exempt. In advanced cases, however, in which the whole choroid is transformed into a dense uniform mass, this region may be densely infiltrated.

The first lymphocytal nodules occur in the stratum of large vessels. They gradually increase in size, and may extend inwards to the middle layer of vessels. The inner layer (chorio-capillaris), however, on account of its elastic tissue, remains for a long time immune, even in very severe cases. Lymphocytes may often be seen lying in dense rows just behind the elastic network, being unable to penetrate it. In some cases they do pass through and reach the lamina vitrea (membrane of Bruch). The lamina is thus seen as a delicate streak on the inner side of the infiltrating tissue. When it in its turn gives way, the cells of the pigment epithelium are open to invasion, and succumb in the usual way. But, as a rule, this does not happen, and although the surface of the choroid be thrown into undulations by the masses of infiltration, the lamina with its epithelium may be quite intact. In this way, the retina (unless otherwise destroyed) tends to be unimpaired in the process of sympathetic infiltration.

Lymphocytes, as usual, constitute the early infiltration. Epithelioid cells appear later and may be difficult to recognise in the dense masses of lymphocytes. With a low power, one should be able to see, in hæmatoxylin and eosin preparations, the reddish areas of epithelioid cells amidst the deep blue of the lymphocytes. Epithelioid cells first appear in the lymphocytal nodes in the large vessel layer. They may outnumber the epithelioid cells in the later stages. Gaps

may be seen in the stroma cells marking the place from which the epithelioid cells have arisen. They occur most abundantly in the posterior segment of the choroid, *i.e.*, where the infiltration is greatest. The infiltration is often seen in the early stages to lie in distinct columns of cells, on account of the elastic laminæ which occur in the choroid. When this elastic tissue gives way in the later stages, the cells are arranged quite irregularly. (The process holds good for invasion of the vessels elsewhere.)

The vessels are affected by the infiltration in the following way. The lymphocytes are at first situated at a distance from the vessel; but gradually they make their way towards the latter, and, having pierced the adventitia, gain entrance to the lumen. The endothelial cells which line the vessel apparently take only a passive part in the process. The invading cells gradually become more and more numerous, the lumen becomes blocked and the endothelial cells destroyed. The mass within the vessel then becomes continuous with the infiltrates outside, and nothing is left to mark the existence of the vessel, except the small bright spot in the centre of the concentrically arranged cells. Even this spot and the concentric arrangement is obliterated in time, and all signs of the pre-existing vessel are lost. The destruction of thin-walled veins is much more rapid than that of the arteries with their thick elastic coats.

Giant cells appear as the epithelioid cells increase; but they may be absent.

(iv) *Tissues outside the Uvea.*—Although the rule is that the inner surface of the choroid remains unimpaired, yet occasionally one finds that it is studded with groups of cells. Single cells of the epithelium proliferate at certain spots and nodules are formed. Lymphocytes gain access to these nodules and masses of characteristic infiltration are formed. The cells may swell up and burst into the subretinal space, and later become organised.

The infiltration, on the other hand, may pass outwards to the suprachoroidea. It may extend also along the

posterior ciliary vessels and nerves, and along the venæ vorticosæ. Typical nodes of infiltration will then be found in the sclera, or outside it. Extensive perforation of the sclera seems to be more common in the sympathising than in the exciting eye.

The retina is seldom affected. It generally retains its position unless detached by severe non-sympathetic inflammation, or by the original violence. There is often a slight infiltration around the veins, but not more than would be present in any ocular disturbance.

The optic nerve is little or not at all affected (very much less so than one would expect were the method of transmission to the other eye, by way of the nerve itself). The changes are merely a slight increase of the nuclei around the vessles.

III. Resolution of the Infiltration.—The infiltration which is characteristic of sympathetic inflammation does not resolve by suppuration or caseation. It organises in such a way that the uvea is gradually transformed into a dense fibrous membrane.

The lymphocytes in the stroma first begin to diminish in number. Epithelioid and giant cells also disappear. As the infiltration decreases there appears between the remaining cells a ground tissue containing large, pale, oval nuclei which have their major axis parallel to the surface of the tissue in which they occur. These, apparently, have at first no protoplasmic body. Later, there grow out from the ends delicate streaks, which are evidently cellular prolongations. These streaks become more pronounced, and finally a dense fibrous structure is produced, closely resembling the sclera.

The origin of the cell elements of this is either directly from the uveal stroma, or from the epithelioid cells, which in their turn were derived from fixed tissue cells of the stroma.

The vessels for the most part have disappeared, but delicate endothelial tubes are seen, and these often take up a

position opposite to that of the original vessels of the part (*e.g.*, in the iris they pass from the deeper layers directly to the surface).

In severe cases the suprachoroidea may show patches of this fibrous tissue, which represent the pre-existing areas of infiltration. When the inflammation has not run a severe course, portions of the uveal stroma may come out of the process unimpaired. For example, in many instances the whole anterior segment of the iris may be left intact, whilst the posterior segment is converted into a dense membrane.

Organisation of the stroma goes on, more or less, at the same time with that of the cyclitic membranes. All the same, one may meet with different stages of the inflammation, in that whilst the infiltration at one part may be fairly recent, another region may show organisation complete.

Sooner or later the infiltration in the stroma, or cyclitic membranes, becomes organised, and the whole uvea becomes a hard mass. Bands of pigment which remain to mark the existence of the retinal epithelial layers project more or less irregularly into the structure. Thus, in the ciliary body, one may see an almost homogeneous connective tissue membrane, poor in nuclei, penetrated by pigmented processes. The unpigmented layer often lies practically unchanged on the inner surface of this membrane.

IV. Plastic Exudation.—This is found mostly in cases of long standing, although even in these it may be absent. Professor Fuchs believes that when it occurs there has been a mixed infection, and that the plastic exudation belongs not to the sympathetic inflammation, but to the accompanying endophthalmitis, which may follow any septic wound of the eyeball. When it occurs it is generally found upon the surface of the ciliary body, and constitutes the "cyclitic membrane." Professor Fuchs draws a very strong line of demarcation between typical sympathetic inflammation, and endophthalmitis. The latter is the condition which occurs

after any septic wound, in which there is no transmission to the other eye. It may vary in its intensity, and, when chronic, is described as fibrino-plastic in its exudations; when acute it may be suppurative.

Sympathetic Inflammation.	Endophthalmitis.
1. The onset is probably never earlier than 14 days after the causal injury.	1. The onset may occur any time after injury.
2. The disease generally runs a protracted course.	2. The course of the disease is generally not much prolonged.
3. There are characteristic cell forms in the infiltration, viz., lymphocytes, epithelioid and giant cells. Polynuclear cells are absent.	3. The cell forms are not constant. In the more chronic cases, lymphocytes are met with. Epithelioid cells are rarely found, and giant cells are absent. Polynuclear cells, especially in the acute forms, are very numerous.
4. There is a characteristic distribution of the infiltration. The uvea is the part essentially affected, and of this the choroid shows the most infiltration. The infiltration, at least in the early stages, is in patches. The stroma is specially involved, and it is the inner segments of iris and ciliary body and the outer layers of choroid that are affected most intensely. The chorio-capillaris is usually exempt.	4. There is nothing characteristic in the distribution of the infiltration. The site of the infiltration is determined by the original injury, as it is in the neighbourhood of the path of infection that the inflammation is most marked. The infiltration is mostly confined to the superficial structures. The stroma, as a rule, becomes affected only secondarily. The infiltration is never patchy, but always diffuse. The choroid is seldom affected, and, when it is, the chorio-capillaris is the part involved.
5. Surface exudations are met with in the late stages, and chiefly consist of lymphocytes. Fibrin is wanting.	5. Exudations invariably occur. They are rich in fibrin and in polynuclear cells. Albuminous fluid bathes the tissues, and this becomes a coagulum in A.C., etc.
6. Organisation takes place, with almost complete destruction of the uveal stroma in severe cases.	6. Organisation of the exudates takes place, often without any involvement of the stroma.

That the picture in a true sympathetic inflammation is not always typical is explained by the fact that one has, in

the exciting eye at least, to deal at the same time with changes which are the result of endophthalmitis. One expects, therefore, that in the sympathising eye, in which there has been no wound to call this forth, one will meet with the manifestations of sympathetic inflammation in the purest form. The sympathising eye always shows a much greater degree of destruction of the tissues than is the case in the exciting eye. It would seem as though the products of endophthalmitis had an inhibitory effect upon the virulence of the etiological factor in sympathetic inflammation.

V. Sympathetic Irritation.—By this is meant the condition of an inflammation in the one eye, accompanied by certain clinical symptoms in the other eye, without any anatomical changes in the latter. These clinical symptoms are chiefly subjective, viz., dimness of vision, pain, and photophobia. Occasionally the objective symptom of ciliary injection may be present. The condition passes off when the first eye is enucleated and does not recur. Professor Fuchs examined 16 cases of irritation, and in all, with one exception, the typical changes of sympathetic inflammation were wanting. The condition of irritation must, therefore, not be confused with that of certain cases of undoubted sympathetic inflammation in which as yet the second eye has shown no clinical signs of disturbance. In the latter case* it is probable that sooner or later the outbreak of sympathetic inflammation will take place on the second eye, whether or not the exciting eye be enucleated.

VI. Serous Traumatic Iritis.—Professor Fuchs describes three cases of this rare condition. The changes met with in them resembled true sympathetic inflammation. The disease is characterised clinically by the presence of keratitis punctata and by the absence of posterior synechiæ. Synechiæ may form at a late stage. The chronicity of the disease is a marked condition. The infiltration is practically confined to the uveal stroma, and in this as well as in the

* *Cf.* Case VI in my Tables (p. 279).

paucity of surface exudations it resembles sympathetic inflammation. It differs from the latter in that it does not tend to pass over to the other eye. Professor Fuchs believes that such cases as these, whilst closely resembling sympathetic inflammation in their anatomical features, differ in etiology very widely, in so far that in the former the disease does not pass over to the other eye, whilst in the sympathetic inflammation transmission invariably occurs.

I have given an account of Professor Fuchs' work in considerable detail, since the picture thereby presented is so very exhaustive.

It will be seen, however, that in it no importance is allotted to the plasma cell. Since in all chronic inflammations of the tissues the plasma cell plays a very large part, I was anxious to find out whether it was a feature of any importance in sympathetic inflammation. With this object I examined cases of sympathetic inflammation and made a quantitative estimation of the plasma cells in each. For comparison with these I also investigated a large number of cases of non-sympathetic inflammation and a few of the more representative of these are given. I shall give a brief description of each case with respect to the points specially laid down by Professor Fuchs. In making his investigation Professor Fuchs selected cases which were typical of sympathetic inflammation. It might be expected, therefore that certain cases of an atypical nature were excluded in this way, and it is interesting to compare cases otherwise selected with his, and find out whether the features mentioned are characteristic in every case.

The method I adopted in making my investigation was this: I first looked up in the records cases which from clinical evidence had been diagnosed as sympathetic inflammation. These cases were derived from a large number of private sources as well as from the practice of the hospital. I obtained in this way 11 cases, in six of which

the eye enucleated was the "exciter," and in five the "sympathiser." I then examined the material histologically to find out in how far the clinical diagnosis would be confirmed by the anatomy. I obtained an additional case when I came to examine the material in a supposed non-sympathetic iridocyclitis. I found that the changes exhibited by this eye were characteristic of sympathetic inflammation. On referring to the subsequent history of the case I found that, although at the time of enucleation absolutely no sign of disturbance had appeared in the other eye, yet the patient nine days later returned to the hospital suffering from well-marked inflammation. I then placed this case amongst my list of sympathetic inflammation and in this way I had in all 12 cases.

In some of the preparations the material had been fixed in formalin (10 per cent.). In the more recent specimens I employed aceto-corrosive solution (corrosive sublimate 6, acetic acid 5, distilled water 100), which is excellent for demonstration of plasma cells. In some of the sections the celloidin method was employed, and in many, paraffin. The specific stain for plasma cells was Pappenheim's solution.* By this the nucleus is stained a greenish purple, and the cell-body bright red. The nucleolus comes out as a very vivid red spot. Polychrome methylene blue I found entirely unsatisfactory.

Although I was not able, in every case, to make preparations in paraffin, and employ the specific stain, yet from constant observation one became familiar with the appearance of the plasma cell, so that even in preparations embedded in celloidin, and stained with hæmatoxylin and eosin, a quantitative estimation could be fairly accurately made. In every case the magnification was the same (Zeiss oil

* The solution is prepared by adding to half a test tube of distilled water two knife-points of pyronin and one knife-point of methyl green. Good rings of individual colour should be produced when the stain is dropped on to filter paper. Differentiation is carried out by absolute alcohol to which a few crystals of resorcin are added. Clearing of paraffin sections is best done by oil of origanum.

imm. 1/12. oc. III) and the average quoted represents the estimation of plasma cells in each field. Only those cells were taken which were distinctive. In some cases where degeneration had set in, or the cells from overcrowding were somewhat indistinct the numbers may be slightly under-estimated.

The Plasma Cell.—For a full description of the plasma cell Mr. Mayou's treatise on inflammation of the conjunctiva, with references given therein, should be consulted. The term was first applied by Waldeyer to the vacuolated and mast cells of healthy connective tissue. Unna used the term to denote pathological cells of the corium, and considered these to be identical with Waldeyer's cells. The term is now universally applied to pathological cells. The cell has an oval rather sac-like protoplasmic body. The nucleus is rather small, rounded, and almost invariably excentric. The chromatin granules are prominent, and are arranged in a typical radiate ("cart-wheel") fashion. Mitosis is frequent, and several nuclei may often be seen in one cell. The cells are distinctly basic in reaction, and thus with Pappenheim's stain the cell-body is red, whilst with hæmatoxylin it is purple. I have practically never found granules in the protoplasm. Atypical cells which are basic in reaction, and contain granular hyaloplasm, are probably a variety of mast cell. Mast cells vary considerably in shape, and are occasionally in outline very much like plasma cells. The nucleus, however, is not as a rule excentric and the granules in the cell body are very marked.*

With regard to the exact origin of plasma cells much dispute has arisen. By some they are regarded as derivatives of connective tissue cells, whilst by others they are said to originate in lymphocytes. Without entering into this controversy I should like to mention that in making a quantitative estimation of plasma cells, it is often very difficult to determine whether certain cells are plasma cells,

* For a description of mast cells, see Macleod, J. M. H., Practical Handbook of the Pathology of the Skin, 1903, pp. 138—140.

lymphocytes, or mast cells. The nucleus is often of the "cart wheel" type in the lymphocyte, and when there is overcrowding of cells, the protoplasmic body does not stand out clearly. Mast cells often have a nucleus like the plasma cell. Whether these three types are only variations of the one original cell, or whether they are morphologically entirely different, is a question that yet remains to be settled. The granules probably indicate a degenerative condition, but I do not agree with Macleod when he says that a basic reaction is degenerative. It is the young fresh cells which take up the basic stain with the greatest avidity. In degenerating cells the protoplasm is often neutrophilic or even acidophilic. I have never been able to trace organisation of a plasma cell into fixed connective tissue. To a certain extent they have powers of migration in that they reach the stroma, but they soon lose this power and it is probable that they degenerate, rather than return to the blood stream. They occur very often in close proximity to vessels, and I have often observed the slender capillaries of the ciliary body for example, tightly packed with plasma cells. I have not noticed pigment in the cell-body, and altogether their *rôle* seems to be somewhat passive. They have been found in abundance in all chronic inflammation of the body; particularly in the so-called infective granulomata (tubercle, syphilis) where they constitute a large proportion of the infiltration. They are generally absent in the acute or suppurative forms, and their presence is generally looked upon as an indication that the tissues in which they occur possess a certain amount of resistance to the toxin which is causing the disease. They occur in the normal conjunctiva, but are called forth in response to the constant stimuli which this very sensitive structure receives. They are absent in the fœtal conjunctiva, since in this case the existence of an irritation is precluded.

A.—Sympathetic Inflammation.

(i) *Exciting Eye.*

Case I.—Patient æt. 6.

History: L. Struck with a toy dart. Wound of cornea extending to limbus.
Iris prolapsed. Traumatic cataract. T –
Prolapsed iris excised soon after injury. Lens escaped at operation; vitreous came up to wound.
L. 6 days later showed doubtful precipitates (K.P.) Capsule in wound. Synechiæ divided.
L. 29 days after injury showed well-marked K.P. None in R.
R. 30 days after injury showed ciliary injection. No K.P.
R. Showed well-marked K.P. 31 days after injury.
L. Excised at this time.

Histology: *Cornea*, wound edges separated by well-organised cellular tissue. Operation wounds for division of synechiæ well healed up.
Canal of Schlemm, *Spaces of Fontana*, much infiltrated.
Iris much infiltrated, especially near wound. Posterior segments more so than anterior. The infiltration is patchy. Giant cells, epithelioid cells and lymphocytes are present. The pars retinalis iridis is broken down at the parts where the infiltration is most intense. Clumps of pigment are scattered throughout the infiltration. The anterior segment of the iris is covered by a thin layer of exudation, the cells of which seem to be continuous with the infiltration cells of the stroma. Clusters of eosinophiles are sometimes seen.
Ciliary body much infiltrated, especially near wound. The pigment layer of the pars retinalis is broken down at parts, and the pigment is heaped up in clumps. Patches of organised tissue are found between the broken down pigment layer and the unpigmented layer (which is mostly intact). Outer zone contains lymphocytes chiefly, but the character-

istic "blue" and "red" areas are not distinct. Exudations are not abundant.

Choroid, patchy lymphocytal infiltration, which is greatest at the posterior pole. It occurs chiefly in the layer of large vessels. The chorio-capillaris is almost quite exempt from infiltration. Pigment epithelium is intact. Giant cells occur. Epithelioid cells are scanty. The infiltration of the choroid is less than that of iris or ciliary body.

Retina, *Optic Nerve* } Unaffected.

Quantitative Estimation of Plasma Cells.

Region ...	Limbus.	Iris.	Ciliary body.	Choroid.	Retina.	Optic nerve at disc.
Average per field	3·16	5·16	5·33	Practically none		1·0

CASE II.—Patient æt. 50.

History: R. Senile cataract extraction.

R. About 6 months later became inflamed. Central ulcer with hypopyon.

L. At same time showed profuse K.P. Vitreous opacities. V = 6/24 $\bar{c}$ correction.

R. Was then excised.

Histology: *Wound* soundly healed and edges in good apposition.

Canal of Schlemm, *Spaces of Fontana* moderately infiltrated (lymphocytes, eosinophiles, and epithelioid cells).

Iris densely infiltrated with lymphocytes and polynuclear cells. The infiltration is rather more marked posteriorly than anteriorly. Epithelioid cells are present. No giant cells. Pigment epithelium entirely destroyed.

Ciliary body infiltrated chiefly in the inner layers. Ciliary muscle also infiltrated. Infiltration consists chiefly of lymphocytes and polynuclear cells. Few

epithelioid cells can be seen. Pars retinalis much destroyed, even the unpigmented layer being at places almost unrecognisable. The dense cyclitic membranes are thus practically continuous with the stroma of the ciliary body.

Choroid almost normal except for a few small patches near the posterior pole. Pigment epithelium has become detached with the retina.

Retina detached. Slight infiltration of the inner layers especially near vessels.

Optic nerve very atrophic.

Polynuclear cells are everywhere abundant. Lymphocytes are numerous. Epithelioid cells occur in small numbers. There are no giant cells. Plasma cells are frequent in iris and ciliary body.

Quantitative Estimation of Plasma Cells.

Region ...	Limbus.	Iris.	Ciliary body.	Choroid.	Retina.	Optic nerve.
Average ...	1·0	13·96	15·16	Practically none		

Case III.—Patient æt. 56.

History: Patient states that one night, 3½ years ago, a sensation of bright light came in R. Next morning R. eye was blind (acute glaucoma).

R. Nine months later became painful and inflamed.

R. and L. operated upon elsewhere at that time. Has not seen well since.

R. on admission was injected. Shallow A.C. No K.P. Both pillars of coloboma in cicatrix. V.R. = No P.L. T – 3.

L. at same time showed enlargement of circumcorneal vessels. Much recent K.P. Pillars of coloboma in wound. V.L. = 3/60. T = + 1.

R. then excised.

Histology: *Wound* extends beyond sclero-corneal margin. Edges separated by a mass of densely cellular organising

tissue. A membrane of similar tissue passes across to the opposite ciliary body. Back of cornea is covered with groups of inflammatory cells.

Iris almost indistinguishably matted up with the membrane in front of eye. Clumps of pigment indicate its former existence. Giant cells and epithelioid cells are numerous, and are often pigmented.

Ciliary body detached. Densely infiltrated, especially in its anterior and inner parts. Ciliary muscle is much opened out and infiltrated. Hæmorrhage has taken place amongst the muscular bundles. Pigment epithelium of pars retinalis is much broken up. Giant cells are very numerous, especially along the "inner zone." Epithelioid cells are universal. The unpigmented epithelium is generally recognisable.

Choroid, patchy infiltration all over, but more marked at posterior pole. Great cellular thickening has occurred at the region of the papilla, and the cells consist chiefly of lymphocytes. The infiltration spreads along the vessels and nerves which penetrate the sclera, passes between the laminæ of the sclera, and along the nerve sheath. Giant and epithelioid cells are numerous. There is profuse choroidal hæmorrhage, and subretinal exudation. Pigment epithelium is much destroyed at parts, and the vitreous membrane is ruptured (probably due to the extravasations).

Retina practically free from infiltration.

Optic nerve somewhat cupped.

The infiltration everywhere consists of lymphocytes, epithelioid and giant cells. Plasma cells are abundant.

Quantitative Estimation of Plasma Cells.

Region ...	Limbus.	Iris and cyclitic membrane.	Ciliary body.	Choroid.	Retina.	Optic nerve.
Average ...	16·4	31·0	13·8	1·33	None	

CASE IV.—Patient æt. 32.

History: L. struck by burst soda-water bottle. Large flap wound. T –

Iris much prolapsed. Prolapse excised. Vitreous escaped at operation. No foreign body discovered by X-rays.

Inflammation ran a severe course. Patient refused enucleation.

R. on readmission, seven weeks later, showed gradually increasing dimness of vision, with ciliary injection. No K.P. Vitreous hazy. V.R = 6/60.

L. then excised.

Histology: Granulomatous mass bulging forwards at the site of wound, which is immediately in front of the ciliary body. The lips of the wound are widely separated by this mass.

Iris—No definite remains of iris in wound, but clumps of pigment indicate its former presence. A dense organising membrane stretches from the mass in wound to the iris of opposite side. Infiltration is greatest in posterior segment of iris. Pigment epithelium of pars retinalis is much broken down, and the infiltration in the iris is continuous with the cyclitic membrane. The infiltration of the iris is patchy, and contains many lymphocytes and epithelioid cells. Giant cells occur, but are not very numerous.

Ciliary body partially detached. Much infiltrated, especially in its inner layers. The muscle also is infiltrated. Pigmented layer of pars retinalis is broken down. The unpigmented layer is generally distinguishable in the infiltration.

Choroid much degenerated. Thickly infiltrated throughout, but especially at posterior pole, and least at the equator. Chiefly lymphocytes and epithelioid cells. A few giant cells. Choriocapillaris is comparatively exempt, but pigment epithelium is raised up by groups of cells occasionally. Membrane of Bruch intact, even at the latter situations.

Retina totally detached, and matted up behind the cyclitic membrane.

Optic nerve atrophic. Slightly infiltrated, especially near its entrance, and in the intervaginal space.

The infiltration is practically of the same type everywhere. Lymphocytes are most abundant. Epithelioid cells are irregularly mixed up with them. Giant cells are occasionally found. Pigmented cells are frequent. Polynuclear cells occur, especially at membrane in pupil. Eosinophiles are numerous. At places (near ciliary body) definite fragmentation of nuclei and caseation occur, giving a resemblance to tubercle. Mast cells occur.

Quantitative Estimation of Plasma Cells.

Region ...	Limbus.	Iris.	Ciliary body and cyclitic membrane.	Choroid.	Retina.	Optic nerve.
Average ...	9·3	8·75	16·75	6·83	—	None

Case V.—Patient æt. 64.

History: R. gunshot wound of ciliary region nine weeks before admission.

L. operated upon for cataract seven years previously. Vision excellent after operation, until ten days before admission, when L. became inflamed. On admission V.L = 6/60 c̄ correction. Photophobia. Fine K.P. Tn.

R. then excised.

Histology: *Wound* passes through region of Ligamentum Pectinatum, and Canal of Schlemm. Edges separated by mass of densely-infiltrated organising tissue.

Spaces of Fontana and filtration angle packed with lymphocytes.

Iris near wound is packed with cells. All the layers are evenly infiltrated. Pigment epithelium broken through, and the two layers of pars retinalis are separated by the infiltration. At parts the posterior

layer also is destroyed, and the iris infiltration becomes continuous with the organising membrane, which lies in front of the lens. On side away from wound iris is infiltrated (a little less densely, however, than the side near wound), and the infiltration is slightly more pronounced in the posterior segment than in the anterior. The cells of the infiltration in the anterior layers pass forwards and form an exudate in the A.C. Lymphocytes, epithelioid, and giant cells are abundant. The epithelioid cells are most numerous in the posterior segment, and at the root of the iris. Polynuclear cells are infrequent.

Ciliary body much infiltrated, especially in the inner layers. The muscle in its anterior part is comparatively free from infiltration. Lymphocytes and plasma cells are numerous. Posteriorly, there are abundant epithelioid and giant cells in the cyclitic exudation. The membrane on inner surface of pars plana is densely organising. The unpigmented layer of the pars retinalis is destroyed at parts.

Choroid, patchy infiltration all over, but densest at posterior pole. Lymphocytes and epithelioid cells are frequent. Giant cells occur. Plasma cells are often met with. Pigment epithelium has been torn away in places, but the chorio-capillaris is practically normal.

Retina—Masses of fibrous tissue lie between retina and choroid, adherent to both. Slight periphlebitis, but otherwise normal.

Optic nerve normal.

Plasma cells are everywhere abundant, but do not constitute the majority of the cells present. Eosinophiles occur frequently.

Quantitative Estimation of Plasma Cells.

Region ...	Limbus.	Iris.	Ciliary body.	Choroid.	Retina.	Optic nerve.
Average ...	9·3	26·08	18·1	6·75	—	—

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CASE VI.—Patient æt. 58.

History: Cataract in both for three years. V.R. = Hand movements only V.L. = 6/12.

R. Extraction with iridectomy.

L. About three weeks later showed marked ciliary blush. No neuritis. No K.P.

R. excised and L. improved.

Three weeks later patient was readmitted suffering from well-marked sympathetic inflammation, which ran a rather severe course.

Histology: Edges of wound separated by densely cellular new-formed tissue which spreads out under the conjunctiva. This tissue contains many large giant cells.

Cellular exudations in A.C., P.C., and vitreous.

Iris, patchy infiltration rather more marked in posterior than in anterior layers. Epithelioid cells occur, but are not well preserved. Extravasations are present. Pigment epithelium mostly intact.

Ciliary body detached. Moderate infiltration of inner layers. Lymphocytes, epithelioid, and plasma cells. Eosinophiles are numerous. No giant cells. Ciliary muscle is atrophic. Unpigmented epithelium has been destroyed at parts by the infiltration.

Choroid detached anteriorly. Moderate patchy infiltration not much more marked at posterior pole. Lymphocytes and epithelioid cells. No giant cells. Extravasations are considerable, and account for the impairment of the inner segment of the choroid. Pigment epithelium at parts is ruptured.

Retina deeply infiltrated in its inner layers, especially round the vessels.

Eosinophiles are everywhere very numerous. Plasma cells are not specially abundant, and show much degeneration. Giant cells are not characteristic in their distribution. Epithelioid cells and lymphocytes are present in all the areas of infiltration.

Quantitative Estimation of Plasma Cells.

Region ...	Limbus.	Iris.	Ciliary body.	Choroid.	Retina.	Optic nerve.
Average ...	2·6	Too much degenerated to be counted	4·16	Practically none		

CASE VII.—Patient æt. 39.

History: R. Cut with barbed wire six weeks before admission.
R. On admission showed profuse K.P., chronic iritis. No P.L. T –
L. On admission showed no injection. No K.P. V.L. = 6/6, J_1.
R. Excised on the following day.
Patient was dismissed well on the fourth day.
Readmitted nine days later. L. said to have become misty lately, and photophobic. L. showed slight ciliary injection, and abundant fresh K.P. Iritis. V.L. = 6/36. Proj. L. good. Improved under treatment, finally gaining V. = 6/9.

Histology: Edges of wound separated by new-formed connective tissue which stretches back also into the vitreous. Wound has become puckered by contraction of this tissue. Epithelioid and giant cells are found in the wound mass.

Iris, absent on side of wound. On opposite side it is much infiltrated. New-formed connective tissue stretches across pupil to wound on opposite side. Infiltration is patchy, and is most marked in the posterior segment. It contains lymphocytes, epithelioid, and plasma cells. Pigment epithelium is torn away in places. There is an exudation on the anterior surface, which is continuous with the cells infiltrating the stroma.

Ciliary body detached and hæmorrhagic. Moderate infiltration, especially in inner aspect. Muscle

slightly infiltrated with lymphocytes, and epithelioid cells. Pigmented layer of pars retinalis distorted. Unpigmented layer at parts beginning to give way.

Choroid detached anteriorly. Intensely infiltrated, especially posteriorly. Infiltration is patchy, and contains lymphocytes and epithelioid cells. Chorio-capillaris is mostly unaffected. Supra-choroidea is infiltrated.

Retina detached, degenerated.

Optic nerve not infiltrated.

The infiltration is everywhere fairly characteristic. Giant cells, epithelioid cells, and lymphocytes abundant.

Quantitative Estimation of Plasma Cells.

Region...	Limbus.	Iris.	Ciliary body.	Choroid.	Retina.	Optic nerve.
Average...	Cells very degenerated	2·0	3·6	Lymphocytes too dense to allow of clear view	Occasi met	onally with

(ii) *Sympathising Eye.*

Case VIII.—Patient æt. 68.

History: R. Extraction with iridectomy for senile cataract. Myopic crescent, and macular choroiditis.

L. Two and a half months later extraction with iridectomy. Iritis, with hypopyon, followed some time afterwards.

R. Seven months later showed intense ciliary injection. K.P., no P.L.

L. Slight ciliary congestion. Old K.P. Vitreous opacities.

R. was excised at this time.

Diagnosis : R. blind and painful from sympathetic inflammation after cataract extraction in L.

Histology: *Cornea* shows delicate connective tissue between epithelium and Bowman's membrane. *Limbus* deeply

infiltrated. Many groups of cells on back of cornea. Fibrinous coagulum in A.C.

Spaces of Fontana contain dense infiltrates.

Iris, moderately patchy infiltration. Chiefly consists of plasma cells. Few lymphocytes or epithelioid cells. Pigmented stroma cells are very abundant. Pars retinalis iridis intact.

Ciliary body—Plasma cells very abundant. Little or no evidence of lymphocytes or epithelioid cells. Chromatophores very marked. Pars retinalis ciliaris mostly intact. Unpigmented epithelium at parts seems to be somewhat disorganised, especially at the regions where there is a slight coagulum on the surface.

Choroid: The tissue is thinned and condensed (probably from myopia). Little or no infiltration. Pigment epitheilum lost near papilla. Its place is taken by connective tissue with scattered pigment.

Retina, atrophic in its inner layers.

Optic Nerve not infiltrated.

There are no giant cells at any part. Many eosinophiles occur. Lymphocytes and epithelioid cells are infrequent. Plasma cells abundant.

Quantitative Estimation of Plasma Cells.

Region ...	Limbus.	Iris.	Ciliary body.	Choroid.	Retina.	Optic nerve at disc only.
Average ...	21·0	20·3	8·83	None		2·83

CASE IX.—Patient æt. 75.

History: L. Extraction with iridectomy for senile cataract. Cyclitis with K.P. followed.

R. Five months later showed ciliary injection. K.P. V.R. = Hand movements only.

L. at this time showed some K.P. No injection. V.L. = 6/36 $\bar{c}$ correction.

R. K.P. gradually disappeared, and patient was discharged much improved.

R. Three weeks later was excised, as inflammation had reappeared.

Histology: *Cornea* normal. Many groups of mononuclear and polynuclear cells on back of cornea, and on front of iris. A few indefinite giant cells are met with in this region. *Limbus* much infiltrated with lymphocytes and plasma cells.

Spaces of Fontana fairly free from infiltration.

Iris much infiltrated in a patchy fashion. Posterior layers not more so than the anterior. Infiltration around the vessels is very dense. Pars retinalis torn away by adhesions. No giant cells. Plasma cells very abundant. Lymphocytes and epithelioid cells infrequent.

Ciliary body—Loose tissue on inner surface of muscle much infiltrated with plasma cells. Muscle itself is infiltrated, but mainly with lymphocytes. Epithelioid cells are rare. No giant cells. Infiltration is dense in pars plana. Organisation has commenced in posterior part. Pigmented epithelium of pars retinalis is absent in places. Unpigmented epithelium proliferating and "dissolved" at places.

Choroid practically free from infiltration, except for a few patches anteriorly.

Retina—Some periphlebitis; otherwise normal.

Optic nerve normal.

Plasma cells are everywhere very abundant. Lymphocytes and epithelioid cells are not very numereus. Giant cells occur in the exudates in A.C. and around lens capsule. Slight coagulum on ciliary body.

Quantitative Estimation of Plasma Cells.

Region ...	Limbus.	Iris.	Ciliary body.	Choroid.	Retina.	Optic nerve.
Average ...	56·0	50·16	37·3	Practically none		

CASE X.—

History: Double iridectomy for glaucoma elsewhere.
L. went wrong at the time and caused sympathetic inflammation of R.
L. was then excised in consequence.
R. has become extremely painful. Severe iridocyclitis.
R. now excised.

Histology: *Wound* well healed and in good apposition. Much infiltrated. Back of cornea covered with cells, which are chiefly polynuclear.

Iris opposite to wound is adherent to cornea for a short distance at periphery. Very densely infiltrated. It is continuous with the membrane passing over to wound. Lymphocytes are present as a rather diffuse infiltration, especially in posterior segment of iris. Patches occur here and there. Epithelioid cells are infrequent. Plasma cells are numerous. Occasional pigmented giant cells occur. Posterior layer of pars retinalis much broken up in places. A mass of highly cellular organised tissue lies between iris and lens capsule, passes into the posterior chamber, and through the fibres of the zonule into the vitreous. A thick layer of degenerating mononuclear and polynuclear cells covers the anterior surface of the iris.

Ciliary body—Some scattered lymphocytes and plasma cells in anterior portion, and on the inner aspect of the posterior portion. Epithelioid cells are scanty. Both layers of the pars retinalis are well preserved.

Choroid mostly empty of blood. A few small patches of lymphocytes in outer layers towards equator; and some very small patches towards papilla. Epithelioid cells are sometimes met with.

Retina normal, except for slight infiltration along vessels.

Optic nerve not much atrophied.

Quantitative Estimation of Plasma Cells.

Region ...	Limbus.	Iris.	Ciliary body.	Choroid.	Retina.	Optic nerve.
Average ...	Lymphocytes too dense to allow of clear view	60·6	39·0	1·0	Practically none	

CASE XI.—Patient æt. 60.

History: R. Sight had been failing for six years. Operation for cataract three months ago elsewhere.

L. Stated to have become affected five weeks after above operation. Steadily became worse.

R. on admission showed conjunctival injection. Old K.P.; iris discoloured and updrawn into coloboma. Pupil filled with lymph. T + 2.

L. On admission showed conjunctival injection, photophobia, K.P., iris discoloured, posterior synechiæ. T + 2. Projection L fairly good.

L. excised, as R. was considered the more hopeful eye.

Histology: Peripheral vessels pass for a short distance under Bowman's membrane. There is a thick uniform layer of cells on the back of the cornea. These are epithelioid, plasma, polynuclear cells, and lymphocytes. Elongated delicate cells, which are probably fibroblasts, are met with. Giant cells occur. Many of the cells in A.C. show pigment granules.

Canal of Schlemm abundantly infiltrated.

Iris covered with exudates, which seem to have come from the underlying stroma. The latter is infiltrated with numerous epithelioid and plasma cells. The infiltration is not much more marked posteriorly than anteriorly. Patches of lymphocytes occur, especially near root of iris. The posterior layer of the pars retinalis is broken down, and the pigment dispersed.

Ciliary body—Infiltration is abundant in the ciliary processes, and in the inner aspect of the pars plana. It consists chiefly of scattered lymphocytes, and plasma cells. Epithelioid cells are scanty. The ciliary muscle is fairly exempt. No giant cells. The pigmented layer of pars retinalis is recognisable throughout as a deep band. The unpigmented layer at parts seems to be undergoing dissolution.

Choroid—Patchy infiltration, which is greatest at posterior pole. Some large patches, however, occur at ora serrata. Lymphocytes are the chief constituents. No giant cells. Epithelioid cells are infrequent. Chorio-capillaris practically normal.

Retina scarcely affected, except at papilla.

Papilla—Delicate web of new-formed tissue containing fibroblasts like those in A.C.

Optic nerve atrophic. Slight infiltration above lamina. Numerous lymphocytes around veins.

Quantitative Estimation of Plasma Cells.

Region ...	Limbus.	Iris.	Ciliary body.	Choroid.	Retina.	Optic nerve.
Average ...	14·6	40·0	42·0	Plasma cells seem degenerated	Occasi met	onally with

CASE XII.—Patient, æt. 68.

History: Senile cataract both. Myopia. V.R. = Fingers at 1 metre. V.L. = Hd. mov. only. Proj. L. good.

R. Extraction with iridectomy.

R. A month after operation showed injection. No K.P. V.R. = 6/18 c̄ correction. No capsule in wound.

R. A month later showed cornea partially opaque. Capsule in wound.

L. At this time intensely injected. Profuse K.P.

over whole of lower part of cornea. Lymph in pupil.

L. then excised.

Histology: *Cornea* normal. Many polynuclear and mononuclear cells on back of cornea, and in A.C. *Limbus* much infiltrated.

Spaces of Fontana packed with cells.

Iris very densely infiltrated. The infiltration is patchy, but is not much more pronounced in posterior than in anterior segment. The cells of the infiltration have begun to penetrate the posterior layer of the pars retinalis, and form a continuous mass with those lying in the posterior chamber. The cells in the anterior segment of the iris also pass out into the A.C. Plasma cells, lymphocytes, and polynuclear cells are abundant. Plasma cells are usually least numerous in the regions where polynuclear cells abound. Giant cells are present, and many are pigmented.

Ciliary body—Moderately infiltrated in its inner aspect. Chiefly with plasma cells. Ciliary muscle little affected. Long spindle-shaped cells (fibroblasts) occur near the ora serrata. The pars retinalis seems to be fairly intact.

Choroid densely infiltrated, especially posteriorly. Infiltration occurs around some of the venæ vorticosæ, and posterior ciliary vessels.

Retina, *Optic nerve* } Normal.

The infiltration is everywhere rich in plasma cells, and lymphocytes. Giant cells occur. Epithelioid cells are not conspicuous.

Quantitative Estimation of Plasma Cells.

Region ...	Limbus.	Iris.	Ciliary body.	Choroid.	Retina.	Optic nerve.
Average ...	22·16	46·6	18·75	40·0	—	—

B. NON-SYMPATHETIC IRIDOCYCLITIS (*i.e.*, ENDOPHTHALMITIS).

History.	Histology.	Quantitative estimation of plasma cells—average for each field.					
		Limbus.	Iris.	Ciliary body.	Choroid.	Retina.	Optic nerve.
CASE I.—Baby. Old iridocyclitis. Recent panophthalmitis. R. began swelling a month before admission. No recent injury alleged. R. excised 2 days after admission. L. remained well, so far as is known.	*Cornea.*—Practically normal. *Iris.*—Much infiltrated. Somewhat patchy, anterior layers slightly less than posterior. New-formed fibrous tissue on posterior surface, which is continuous with stroma, pars retinalis being entirely broken down. *Ciliary body.*—Deeply infiltrated with lymphocytes and polynuclear cells. Pigment epithelium destroyed. Unpigmented epithelium also unrecognisable. *Vitreous* and *Choroid.*—Deeply infiltrated with lymphocytes and polynuclear cells. *Retina.*—Detached, much infiltrated, and destroyed. *Optic nerve.*—Atrophic. Infiltrated, chiefly with polynuclear cells. Exudations on surface of uvea. Infiltrates contain lymphocytes, polynuclear cells, and a few epithelioid cells. No giant cells.	1·33	3·66	0·5	4·3	Practically none	

CASE II.—Patient æt. 55. Old iridocyclitis. 3 years ago was an outpatient for iridocyclitis in R. 5 months before readmission had a fall on head, when R. eye became inflamed. No K.P. Pupil occluded. No P.L. R. then excised. L. showed no affection at any time.	*Cornea.*—Vascular at periphery. Exudates on posterior surface. *Iris.*—Infiltration not patchy, chiefly mononuclear cells. Pupil filled with firm connective tissue. The two layers of the pars retinalis are widely separated by connective tissue. *Ciliary body.*—Detached. Unpigmented layer of pars retinalis unrecognisable. *Choroid.*—Infiltrated. Subretinal exudate. Pigment epithelium degenerated. Posterior segments thickened and in folds. Connective tissue in suprachoroidea. *Vitreous.*—Full of dense connective tissue. *Retina.*—Detached, degenerated. *Optic nerve.*—Atrophic. Plasma cells fairly abundant. No giant cells. ? Epithelioid cells.	3·16	8·3	8·16	Practically none
CASE III.—Patient æt. 27. Dislocated lens. Iridocyclitis. 7 years ago was an outpatient, with iridocyclitis and cataract R. No history of injury, but patient is a boxer. Periodical attacks of pain in R. since then.	*Cornea.*—Normal. A.C. full of blood. Lens dislocated, lies upon ciliary processes. *Iris.*—Stroma condensed. Slight infiltration of lymphocytes; not more marked in posterior segment. Pigment epithelium broken down. The layers of pars retinalis are separated by blood. Infiltration is somewhat patchy. Vessels unimpaired. No giant cells. *Ciliary body.*—Exudates on pars plana. In-	2·0	4·83	—	Practically none

B. NON-SYMPATHETIC IRIDOCYCLITIS (*i.e.*, ENDOPHTHALMITIS)—*continued.*

History.	Histology.	Quantitative estimation of plasma cells—average for each field.					
		Limbus.	Iris.	Ciliary body.	Choroid.	Retina.	Optic nerve.
CASE III—*continued.* R. on admission now shows ciliary injection. Cornea hazy. A.C. almost full of blood. R. shortly after was excised. No affection of L. at any time.	filtration of stroma very inconsiderable. Muscle slightly infiltrated. Unpigmented epithelium of pars retinalis proliferating in places. *Choroid.*—Detached anteriorly. Some lymphocytes posteriorly. No giant cells. No epithelioid cells. *Retina.*—Detached. Subretinal hæmorrhage. *Optic nerve.*—Atrophic.						
CASE IV.—Patient æt. 26. L., old iridocyclitis. Choroido-retinitis. Myopia. Attended as an out-patient 9 months before admission. L. on admission flushes on examination. Old and recent K.P. Iris discoloured. No P.L.	*Cornea.*—Œdematous. Leucocytes numerous in meshes of ligamentum pectinatum. *Iris.*—Stroma cellular. Infiltration is diffuse. Plasma cells are numerous, especially in the posterior segment. *Ciliary body.*—Detached, and loosened in structure. Pigmented layer of pars retinalis much distorted. Unpigmented layer also affected. Delicate but fully organised membrane over the inner surface.	1·0	10·2	17·3	1·7	8·0	—

L. excised next day. No affection of R. at any time.	*Choroid.*—Detached anteriorly. Much thickened and thrown into folds posteriorly. Lymphocytes and plasma cells are numerous. Infiltration is not greater at posterior pole. Pigment epithelium impaired. Choriocapillaris infiltrated. *Retina.*—Much infiltrated. Adherent to choroid. Lymphocytes and plasma cells. *Optic nerve.*—Normal. Infiltrations consist of plasma cells and lymphocytes. No epithelioid cells. No giant cells. Exudations on surface of ciliary body are abundant.						
Case V.—Patient æt. 33. L. struck with chip of red hot iron. No f.b. (X-ray). Prolapse iris. L., 3 weeks after injury, showed injection and lachrymation. No K.P. Readmitted 18 days later. L. irritable, photophobia. No K.P. L. 12 days later showed K.P. None in R. L. enucleated 3 days afterwards.	*Cornea.*—Wound firmly healed, with downgrowth of epithelium in its superficial layers. *Limbus* much infiltrated around deep vessels. *Episclera* also infiltrated. Eosinophiles and lymphocytes numerous. No epithelioid cells. *Iris.*—Adherent to wound by superficial layers. Tissue much pulled out anteriorly, but posterior layers hardly at all affected. Infiltration around canal of Schlemm. Exudation on anterior surface. Cells near root show delicate fibrillations at their extremities. Lymphocytes occur in small numbers. Not specially infiltrated in posterior layers. Mast cells, but few plasma cells.	5·0	4·0	7·3 in patches	None	None	None

B. NON-SYMPATHETIC IRIDOCYCLITIS (*i.e.*, ENDOPHTHALMITIS)—*continued.*

History.	Histology.	Quantitative estimation of plasma cells—average for each field.					
		Limbus.	Iris.	Ciliary body.	Choroid.	Retina.	Optic nerve.
CASE V—*continued.* No affection of R. at any time.	*Ciliary body.*—Muscle slightly infiltrated. Occasional epithelioid cells in "inner zone" cells show degeneration. Patches of plasma cells in the anterior segment near vessels. *Choroid.*—Congested. Pigment epithelium torn away. Disturbance of pigment in stroma cells. Chorio-capillaris mostly unaffected. Here and there a small node of lymphocytes and eosinophiles are seen in the outer layers. *Retina.*—Detached slightly, otherwise normal. *Optic nerve.*—Normal. Exudations are everywhere scanty.						
CASE VI.—Patient æt. 20. L. struck with piece of wood 3 days before admission. Prolapse of iris. Inflammation	*Cornea.*—Large wound, edges separated by granulation tissue. Stroma vascular and œdematous. *Limbus* much infiltrated, chiefly with plasma cells and lymphocytes, especially in the deeper layers.	Everywhere plasma cells very numerous, specific stain not available, and sections rather old to make an accurate estimation.					

ran a fairly severe course. L. excised 4 weeks later. No affection of R. at any time.	*Iris.*—Indistinguishably matted up with the mass which fills in the wound, and is continued backwards to lens. Organisation is proceeding, but is not very advanced. Giant cells and a few foreign bodies (? hairs) lie in the deeper layers. *Ciliary body.*—Mostly recognisable as such. Infiltrated, especially in pars plana. Leucocytes lie among the unpigmented cells of pars retinalis. Plasma cells in muscle and pars plana. *Choroid.*—Only very slightly infiltrated anteriorly. Pigment epithelium broken away. *Retina.*—Infiltrated along vessels. Detached. No epithelioid cells at any part. Exudations are everywhere abundant. Plasma cells very numerous.	
Case VII.—Patient æt. 42. L. struck with piece of steel. Iris adherent to wound. No A.C. No f.b. (X-rays). Inflammation ran a severe course L. 4 weeks later showed K.P. R. normal. L. excised a few days later.	*Cornea.*—Wound firmly healed. Some delicate new-formed tissue between epithelium and Bowman's membrane, near wound. Edges of wound in deeper parts separated by a dense avascular fibrous tissue. This mass stretches out behind iris, and is attached to remains of lens. *Limbus* shows occasional mast cells. *Iris.*—Slightly infiltrated. The infiltration consists of scattered lymphocytes. Mast cells occur. *Ciliary body.*—Slight exudation. Stroma not	Practically no plasma cells.

B. Non-sympathetic Iridocyclitis (*i.e.*, Endophthalmitis)—*continued.*

History.	Histology.	Quantitative estimation of plasma cells—average for each field.					
		Limbus.	Iris.	Ciliary body.	Choroid.	Retina.	Optic nerve.
Case VII—*continued.* R. had not shown any sympathetic inflammation 2 months after excision L.	infiltrated. Pars retinalis not much impaired. *Choroid.*—Not infiltrated, but a little congested. *Retina* and *Optic nerve.*—Normal. Practically no plasma cells occur. Mast cells are frequent.						
Case VIII. — Cat's eye showing iridocyclitis. Followed upon excision of one half sympathetic nerve in neck, two months previously.	*Cornea.*—Vessels run in from sclero corneal margin for some distance. Stroma scarcely infiltrated. *Iris.*—Layer of inflammatory cells on anterior surface, in pupil, and in angle of A.C. Stroma much infiltrated with plasma cells, especially around the vessels. *Ciliary body.*—Much distorted by organised membranes in vitreous. Epithelium detached, but unpigmented layer generally recognisable. Necrosis has occurred at some parts.	16·0	25·5	4·0	—	—	—

	Choroid and *Retina*.— Indistinguishably matted up in a mass of round cell infiltration (lymphocytes and epithelioid cells) which extends from papilla to ora serrata. Plasma cells not abundant here. *Optic nerve*.—Slight infiltration in sheaths. There are no giant cells at any part. Mast cells occur. Lymphocytes and epithelioid cells are found. Plasma cells are abundant.					
CASE IX.—Patient æt. 47. L. iridocyclitis and secondary glaucoma. Out-patient for last 9 years with this complaint. L. on admission, intense ciliary congestion. Fine K.P. Iris discoloured, much vascularised V.L = no P.L. R. normal. L. then excised. R. remained unaffected.	*Cornea*.—Fairly normal. *Iris*.—Atrophic, much condensed, infiltrated. *Ciliary body*.—Atrophic. Pars retinalis destroyed and its layers not recognisable. *Choroid*.—Degenerated. Over macular region there is a thick layer of very dense fibrous tissue, with scattered pigment in its meshes. Pigment epithelium lies on its inner surface. Pigment cells are blown up. *Retina*.—Detached, degenerated. *Optic nerve*.—Atrophic. Plasma cells are scanty. No giant cells. Lymphocytes and polynuclear cells are abundant. A few epithelioid cells. Mast cells occasionally occur.	Practically no plasma cells				
CASE X.—Patient æt. 69. Iridocyclitis following L. cataract extraction 4 months previously.	*Cornea*.—Œdematous. *Limbus* much infiltrated with lymphocytes and plasma cells. A.C. full of hæmorrhage and exudation. *Iris*.—Dense mass of new-formed fibrous	37·0	23·3	5·8	None	8·3	—

B. NON-SYMPATHETIC IRIDOCYCLITIS (*i.e.*, ENDOPHTHALMITIS)—*continued.*

History.	Histology.	Quantitative estimation of plasma cells—average for each field.					
		Limbus.	Iris.	Ciliary body.	Choroid.	Retina.	Optic nerve.
CASE X.—*continued.* R. was also cataractous. No inflammation occurred in R. as result of operation in L.	tissue on surface, continuous with mass in pupil and A.C. Intense infiltration of stroma with lymphocytes. Not more marked in posterior than in anterior segment. Pars retinalis broken down, and infiltration in stroma becomes continuous with the exudations in posterior chamber. These exudations show organisation. Giant cells are frequent. *Ciliary body.*—Practically no infiltration of stroma. Pars retinalis much broken down at side of wound. *Choroid.*—Free from infiltration. *Retina.*—Much infiltrated in region of vessels. Lymphocytes chiefly. *Optic nerve*—Normal. Plasma cells abundant. Lymphocytes are frequent. No definite epithelioid cells. Giant cells well marked.						

Conclusions.

When an examination of the preceding cases is made, it will be seen that the results of my investigations are on the whole confirmatory of the description of sympathetic inflammation given by Professor Fuchs. The exceptions will be found to lie chiefly in the examples of the inflammation as it occurs in the sympathising eye. Since this was not included in Professor Fuchs' description, the divergence in our results is, therefore, only an apparent one. In two of my five cases there was an operative wound in the sympathising eye, but in the other three no injury had taken place. In the three latter cases (ix, xi, xii), therefore, one expects to find the condition of sympathetic inflammation in an uncomplicated form. As no essential difference seemed to hold between the two cases in which the operative wound existed and the other three, a summary of the features may be given for the cases as a whole.

With regard to the cell-forms in the infiltration, epithelioid cells were infrequent throughout. Lymphocytes were abundant in four of the cases. Giant cells were absent only in one case. Plasma cells were exceptionally numerous in all the cases. Delicate fibroblasts were found in the exudates of several. With regard to the distribution of the cellular elements, the infiltration was usually in patches. In many cases the patches consisted chiefly of plasma cells. The iris and ciliary body were usually affected, and in the usual situations. The choroid was densely infiltrated in two cases. In the other three it was practically free from infiltration. Slight exudations were present, as a rule, over the iris and ciliary body. The infiltration seemed to be less rich in cells than is the case in the exciting eye. The unpigmented epithelium of the pars retinalis of the ciliary body seemed in some cases to be considerably disorganised, and there was an albuminous exudate which streamed away from its surface into the vitreous. The appearance presented by these eyes was that of tissues which were dissolving under

the action of some powerful toxin. The infiltrations were not so plentiful as in the case of the exciting eye, and consisted mainly of plasma cells particularly in the region of the uveal vessels.

With regard to the anatomical conditions which exist in the exciting eye, it will be seen that the features indicated by Professor Fuchs as characteristic are present practically in every case. This is all the more striking when it is borne in mind that our investigations were approached from opposite starting points. Professor Fuchs selected 35 cases in which the anatomical features were characteristic, and verified this by an appeal to the clinical history. I selected my cases chiefly from the clinical diagnosis in order to observe whether there is an anatomical picture to correspond. From a consideration of the few points of difference in our results, one feels that although, as a rule, the anatomical and clinical conditions are in strict agreement, yet atypical cases occur, in which one has to fall back upon the anatomy for one's diagnosis, and *vice versâ*. The thin line that separates the condition of "irritation" from that of inflammation is an indication of how the clinician may be baffled in making a definite diagnosis: and the cases of well-marked histological features in which all clinical symptoms were wanting still further complicate the problem of sympathetic disease. With regard to the cell-forms in my seven cases of sympathetic inflammation of the exciting eye, lymphocytes, epithelioid, and plasma cells were found in all. Giant cells occurred in all except Cases ii and viii (out of my 12 cases giant cells occurred 10 times). With regard to the characteristic distribution of the infiltrates, I found that in most cases it was typical for each region involved. With regard, however, to its intensity in the various positions of the uvea, the choroid in my cases was not particularly prominent. In five cases the anterior portion of the uvea was more involved than the choroid (in four of these the choroid was practically exempt); in seven cases the choroid was much infiltrated, but only in two of these to a greater

extent than the iris and ciliary body. The infiltration was usually of a patchy nature, except in the severe cases, where it was diffuse. I have not always been able to distinguish the definite "blue and red" areas in hæmatoxylin-and-eosin specimens described by Professor Fuchs.

That the presence of the plasma cell is a well-marked, if not an essential, feature in the anatomical picture of sympathetic inflammation will, I think, receive some credence when the quantitative estimation in the above cases has been considered. That the plasma cell did not receive more recognition from Professor Fuchs may be ascribed to the fact that most of his material had been fixed in Müller's fluid—a reason which he himself gives for the manifest infrequency of mast cells in his preparations. It is possible also that under the term epithelioid, Professor Fuchs may have included the plasma cell, especially since his diagnosis would not be made by aid of Pappenheim's specific stain. Then again, Professor Fuchs did not investigate the anatomy of the sympathising eye. It is here that the plasma cell is seen in its greatest abundance—a fact which still further emphasises its existence as a characteristic feature of sympathetic inflammation in its uncomplicated form.

From a comparison of the examples of non-sympathetic inflammation which are given above, it will be seen that the numbers of plasma cells are on the whole greater in the sympathetic than in the non-sympathetic cases. The two exceptions amongst the latter (Case vi and Case x), in which the numbers are fairly large, may be looked upon as atypical, since in one we are dealing with the eye of the cat, and in the other the occurrence of giant cells at least indicates another feature shared in common with sympathetic inflammation. Incidentally, I should like to draw attention to the fact that in all my cases, as has been so constantly observed by others, the optic nerve, ciliary vessels and nerves were practically free from any infiltration whatever.

In conclusion, I wish to express my indebtedness to the surgical staff of the hospital for the use of the pathological

material. I have also to thank the curator, Mr. George Coats, for his constant supervision of my work, and many valuable suggestions.

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THE FATE OF EYES THAT HAVE BEEN SUBMITTED TO THE OPERATION OF EXTRACTION OF A FOREIGN BODY BY THE ELECTRO-MAGNET.

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Between the years 1901 and 1905 inclusive, 118 cases of iron or steel foreign bodies within the eye were submitted to the operation of magnet extraction. As many of these cases as possible have been traced and examined, and it is the object of this paper, with these cases as material, to add to our knowledge of the ultimate fate of such injured eyes.

Several methods of operating are in use at Moorfields, so that there will be an opportunity of comparing the results obtained by these various plans.

The cases that follow show that the commonest procedure is to drag forward the foreign body into the anterior chamber, or disentangle it, by means of Haab's giant magnet and then to make an incision in the cornea with a keratome or broad needle in a position diametrically opposite the foreign body, and half-way between the corneo-scleral margin, and the edge of the pupil, and then to introduce the terminal of the small magnet or magnetised spud, and so remove the foreign body. This method is not only used for cases in which the foreign body is situated in the vitreous, but also to release those embedded in the iris or lens.

Another plan, after drawing the foreign body forward with the Haab magnet, is to make a *peripheral* incision with a keratome, and in this way introduce the terminal of the small magnet. Now, there are three serious objections to this procedure :—

(1) The possibility of prolapse of the iris and the need of an iridectomy, which is practically a certainty in

those cases in which the foreign body is entangled in the iris.

(2) The possibility of subsequent adhesion of the iris at its base to the wound, which it is almost impossible to release.

(3) The bruising of the tissues and greater difficulty in operating, due to the fact that, compared with the range offered by a corneal incision, a much smaller peripheral incision is used from fear of a prolapse of iris; that although a very peripheral incision is planned, it is seldom so peripheral as anticipated, and consequently a ridge is left, formed by the lower lip of the incision, against which the foreign body can easily be caught.

A third method applies only to foreign bodies situated in the vitreous, and that is the plan of puncturing the sclerotic radially (to avoid injuring blood-vessels as far as possible), and then to introduce the terminal of the small magnet immediately over the situation of the foreign body. This method is only applicable after an examination and localisation by means of the X-rays. Mackenzie Davidson's method is that in use at Moorfields, and its extreme accuracy has often been tested in those cases in which the foreign body could be seen in the anterior chamber or lens, or in which a scleral incision has revealed the foreign body immediately beneath the point of the knife; also by the pathological examination of excised globes in which the foreign body remained. In no case at Moorfields has the plan been adopted of introducing the magnet through the sclerotic diametrically opposite the foreign body.

Other points of importance are the site of entry of the foreign body, the path it pursues within the globe, and its final resting-place. These facts affect the prognosis as much as the method adopted for its removal.

With a view to ascertaining the prognosis of these injuries as many cases as possible have been traced and

examined at a period of not less than one year after the extraction (or attempted extraction) of the foreign body. A few cases are included in which the final condition was already known.

Forty-two cases have been carefully examined either by me, or by different ophthalmic surgeons in the country at my request, in those cases in which the patients lived at too great a distance from London to present themselves at Moorfields.

In thirty-eight cases letters requesting them to come and be examined have been returned through the Dead Letter Office. This, of course, was due to the fact that the majority of accidents of this nature occur in patients of the labouring class, who are constantly moving about the country.

In thirty-eight cases the eye injured was excised.

In sixty-seven cases the left eye was the one injured, and in forty-nine the right eye. This discrepancy is explained by the fact that the majority of patients are right-handed, and the chisel and hammer, from which the majority of intraocular foreign bodies come, are used so that the left eye is nearer the area of danger, and also that the nose must be a protection to the right eye.

In only one case was the patient a woman.

In two cases the other eye had been previously excised for a similar accident.

In six cases operative procedures were of no avail in removing the foreign body.

Case 1.—William D., aged 18, under Mr. Lang, was admitted January 23, 1888. The left eye had been injured three days before by a chip from a chisel from another bench at his work. The eye was much congested, there was a wound of the cornea below, iritis, hypopyon. V. = perception of light. T. low.

A peripheral section was made down and in, and the hypopyon evacuated. The small magnet was introduced with no effect. Iridectomy was performed, and the magnet introduced

into the vitreous without effect. Vitreous escaped, the lens was partially scooped during which more vitreous was lost.

14.ii.88. Eye quiet. V. = hand movements.

1.v.88. Tension +1.

7.v.88. Partial curette evacuation.

17.v.88. Needling.

7.vi.88. Iridotomy.

July, 1888. Vision with glass = 6/9 and J. 1.

23.v.03. V. = 6/9 and J. 1.

25.vii.07. Left eye. Quiet. Cornea clear, but for dense leucoma adherens below. The whole iris is drawn down to it. Much very fine K.P. Good iridotomy opening. Optic disc normal. V. with glass = 6/9. J. 1.

The right eye had been excised in 1885 for a similar accident. In 1899 the patient was X-rayed by Dr. Mackenzie Davidson, and a foreign body could be seen in the negatives in the left eye below in the region of the ciliary processes.

Case 2.—William S., aged 37, under Mr. Collins. Fifteen months before admission—April 1, 1901—the left eye was struck by a flying chip whilst he was picking the road. There was a small scar in the outer part of the cornea; the lens was clear, and there was much vitreous opacity. The foreign body could be seen just to the outer side of the optic disc in the retina. The X-rays confirmed this. The Haab magnet, and the small magnet introduced through a scleral incision, failed to remove the foreign body.

30.viii.07. Left eye. Quiet. No injection. Cornea clear. Many finely pigmented spots of K.P. Good anterior chamber. Iris a little atrophic. One posterior synechia. Lens completely opaque and swollen. T. low. V. = no perception of light.

Right eye. Normal. V. = 6/9. $\bar{c}$ +0·75 D cyl. Ax. H. = 6/5. Adde +1 D sph. = J. 1.

Case 3.—William W., aged 17, under Mr. Spicer. The right eye was injured seven months before admission, July 12, 1901. There was a minute scar in the outer part of the cornea, and a partially absorbed cataract. V. = hand movements. Good projection. The foreign body was localised by the X-rays in the lens.

17.vii.01. The foreign body was removed by the small magnet. Curette evacuation.

23.vii.01. V. with glass 6/6. J. 1.

14.x.07. Right eye. Quiet. Minute corneal scar above. Pupil dilates well. Fine, bright capsular membrane. No vitreous opacity. Fundus n. T. n. V. $\bar{c}\,\frac{+9\text{ D sph.}}{+1\text{ D cyl. Ax. V.}}$ 6/6.

Adde +4 D sph. = J. 1.

Left eye. Normal. V. 6/9. With −0·5 D sph. = 6/6. J. 1.

Case 4.—Robert H., aged 25, under Mr. Fisher. The day before admission—August 14, 1901—whilst passing a man who was chipping, the left eye was injured by a flying fragment. There was a horizontal wound of the cornea and traumatic cataract.

17.viii.01. Hypopyon present. The Haab magnet drew the foreign body forward, and it was removed by the small magnet. Iridectomy was then performed.

11.ix.01. Division of anterior synechia.

6.vi.03. Field quite good. Quiet. No K.P. Pupil bound down considerably. Remains of lens matter.

13.vi.03. Needling.

16.vi.03. V. $\bar{c}\,\frac{+10\text{ D sph.}}{+1\text{ D cyl. Ax. 60° down and out}}$ 6/24.

8.viii.03. Eye quiet.

Case 5.—Henry B., aged 18, under Mr. Fisher. Nine weeks before admission—September 3, 1901—the left eye was injured whilst he was rivetting. There was a small central scar of the cornea, no K.P., and a traumatic cataract in which a foreign body could be seen. The X-rays failed to show its presence.

4.ix.01. The Haab magnet released the foreign body, which was removed by the small magnet through a corneal incision.

6.ix.01. Lens dislocated.

7.ix.01. Scoop extraction.

24.ix.01. Vision with glass 6/12 and J. 1.

14.ix.07. Left eye. Quiet. Small central corneal scar. No K.P. Coloboma upwards. Inner pillar and capsule adherent

to back of cornea. Fine crinkled capsule. No vitreous opacity. Fundus normal. V. $\bar{c}$ +10 D sph. = 6/18; +14 D sph. = J. 2.

Right eye. Normal. V. = 6/6 and J. 1.

Case 6.—H. B., aged 36, under Mr. Silcock. Twelve hours before admission—October 10, 1901—the left eye was injured whilst he was chipping a steel plate. The cornea was wounded down and in, and a small foreign body could be seen lying on the iris.

The Haab magnet failed to dislodge the fragment, but the small magnet, introduced through a peripheral section, was successful. Iridectomy was then performed.

22.x.01. A small spot of opacity in posterior part of the lens.

1.i.02. A little lens opacity down and out. The remainder of the lens clear.

V. = 6/24. $\bar{c}$ $\left.\begin{array}{l} -0{\cdot}5 \text{ D sph.} \\ -1 \text{ D cyl. Ax. } 45^\circ \text{ down and out} \end{array}\right\}$ 6/9.

29.xi.02. Eye in same condition as at previous note.

Case 7.—James P., aged 37, under Mr. Collins. The day before admission, November, 20, 1901, whilst he was chipping a stone the left eye was injured. There was a wound in the inner part of the cornea, and a foreign body could be seen lying on the iris. The lens was clear.

21.xi.01. The foreign body was removed by the small magnet.

2.xii.01. Left vision = 6/18.

20.viii.07. Left eye. Quiet, and but for corneal scar, normal. V. = 6/5.

Right eye. Normal. V. = 6/5.

Case 8.—William S., under Mr. Lawford. Five weeks before admission, November 29, 1901, the right eye was injured by a chip from a hammer. There was a corneal scar down and in, and a track of opacity running through the lens. Nothing abnormal was seen with the ophthalmoscope. V. = 6/18. By the X-rays a foreign body was localised in the vitreous.

30.xi.01. The Haab magnet not successful.

4.xi.01. The small magnet introduced over the spot of localisation unsuccessful.

18.xi.01. Further attempt with the small magnet without success.

1.xi.02. Vision = 6/18. Vitreous hæmorrhages.

CASE 9.—Henry S., aged 18, under Mr. Spicer. Five weeks before admission, January 8, 1902, the right eye was injured whilst he was drilling. There was a small wound just outside the corneo-scleral margin on the inner side, and traumatic cataract. An X-ray examination localised a foreign body in the bottom of the vitreous.

14.1.02. The foreign body was drawn into the anterior chamber by the Haab magnet, and it was removed through a corneal incision by the small magnet.

21.vi.02. Needling.

20.viii.07. Right eye. Deviates outwards. Small pigmented scar 3 mm. outside cornea. No K.P. Pupil active, regular. Iris tremulous. Fine crinkled capsular membrane. Fine vitreous opacities. Fundus normal.

$$\text{V. } \bar{c} \left.\begin{array}{l} +10{\cdot}5 \text{ D sph.} \\ \hline +1 \text{ D cyl. Ax. } 50^\circ \text{ down and in} \end{array}\right\} 6/12.$$

Left eye. Normal. V. = 6/6 J. 1.

CASE 10.—William L., aged 15, under Mr. Collins. A year before admission, June 4, 1902, the left eye was injured by a flying chip from a sledge hammer. There was a wound of the sclerotic below. Partial dislocation of the lens and retinitis proliferans in which a foreign body could be seen. The Haab magnet and the small magnet introduced through a scleral incision did not remove the foreign body.

17.xi.04. Left eye. Quite blind. Pupil obstructed by degenerated lens and capsule. Deviates inward.

Right eye. Normal.

CASE 11.—Henry R., aged 45, under Mr. Spicer, was admitted July 26, 1902. His left eye had been injured by a flying fragment. There was a vertical wound of the corneo-scleral margin. A large foreign body was removed by the Haab magnet. The prolapsed iris was removed.

13.viii.02. Left vision = 6/24. Blood and cicatricial tissue extending from scleral wound into the vitreous.

5.viii.07. Left eye. Quiet, but flushes a little when examined. Two to three conjunctival vessels passing to outer side of corneal margin. No K.P. Fibrous tissue extending from wound into vitreous. No gross change in fundus, T. low n. V. = 6/36.

Right eye. Normal. V. = 6/6 J. 1.

Case 12.—Albert D., aged 35, under Mr. Morton. Eight days before admission, December 16, 1902, the left eye was injured whilst he was working at an anvil. There was a scar of the upper part of the cornea, the pupil well dilated, and commencing cataract.

A small foreign body was localised in the vitreous by the X-rays. The foreign body was removed through a corneal incision below, by the Haab magnet.

30.xii.02. Lens becoming opaque.

Notes from the hospital at Ipswich :—

22.vii.04. V. $\bar{c}$ +12 D sph. = 6/12.

29.xii.04. Right vision = 6/6. Left vision, hand movements.

29.x.07. Mr. Sinclair, of Ipswich, has kindly sent me a note of the present state.

Right eye. Quiet. T. n. Clear gap in capsular membrane. V. $\bar{c}$ +12 D sph. = 6/12.

Left eye. Normal. V. = 6/6.

The right eye has been needled twice, and has had a curette evacuation.

Case 13.—Charles M., aged 22, under Mr. Lawford. A month before admission, December 18, 1902, the left eye was struck by a flying chip whilst he was turning a steel crank. There was a small central scar of the cornea, no iritis, and a traumatic cataract in which a metallic body was seen.

20.xii.02. The foreign body was removed by the small magnet through a corneal incision above.

24.xii.02. Curette evacuation.

14.i.02. V. $\bar{c}$ +13 D sph. = 6/18.

24.i.02. Pupil nearly clear. T. n.

14.x.07. Left eye. Quiet. No K.P. Small central corneal scar. Good anterior chamber. Pupil active and regular. Fine

capsular membrane. Fundus and vitreous normal. V. c +16 D sph. = 6/36.

Right eye. Normal. V. = 6/6. J. 1.

Case 14.—John P., aged 31, under Mr. Collins. Six days previous to admission, December 29, 1902, the left eye was struck by a flying chip of metal. There was a scar just to the inner side of the centre of the cornea, and a small foreign body was seen lying on the iris.

29.xii.02. The foreign body was released by the Haab magnet, and removed by the small magnet through a peripheral incision.

31.xii.02. Right vision with glass = 6/6.

27.viii.07. Left eye. Quiet. Corneal scar as above. No K.P. Pupil dilates well. Lens clear. No vitreous opacity. Fundus normal. V. = 6/12. With +0·5 D sph. = 6/6. With +1 D sph. = J. 1.

Right eye. Normal. V. 6/9. With +0·5 D sph. = 6/6. J. 1.

Case 15.—Herbert C., under Mr. Silcock. On the day of admission, February 12, 1903, a flying fragment struck his left eye. There was a wound of the cornea, down and in, in which the iris was caught, and traumatic cataract. The foreign body, 14 mm. in length, was removed by means of the Haab and small magnets. Iridectomy was then performed.

5.iii.03. Lens partially opaque. V. = 3/60.

22.vi.07. Left eye. Quiet. No injection. Cornea clear but for a small dense leucoma adherens down and in. No K.P. Good anterior chamber. Iris a little atrophic, and an imperfect coloboma down and in adherent to the cornea. Dense opaque capsular membrane, and lens remains. T. n. V. = fingers at 1 metre. Projection good.

Right eye. Normal but for several small scars in the cornea. V. = 6/6. J. 1.

Case 16.—Charles B., aged 22, under Mr. Tay. On the day of admission February 16, 1903, whilst chipping a bicycle frame a fragment of steel flew into his right eye. There was a wound of the cornea up and out, a notch in the pupillary edge of the iris, and traumatic cataract. The foreign body was drawn

forward into the anterior chamber with the Haab magnet and removed by the small magnet.

25.ii.03. R.V. = 4/60.

25.xi.03. R.V. = hand movements. Projection good.

30.xi.03. Needling.

7.xii.03. Curette evacuation.

19.vi.07. Right eye. Quiet. No injection. Scar in cornea and notch in iris, as above. Pupil dilates well. No synechiæ. Clear gap in capsular membrane. No vitreous opacity. Fundus normal. T. n. V. $\bar{c}$ + 13·5 D sph. = 6/5. + 17 D sph. = J. 1.

Left eye. Normal. V. = 6/5. J. 1.

Case 17.—George T., aged 38, under Mr. Collins. On the day of admission, August 19, 1903, the left eye was injured whilst he was chipping. There was a wound of the cornea up and out. No iritis. The lens clear. The X-rays proved the presence of a foreign body in the globe.

20.viii.03. The Haab magnet drew the foreign body forward and it was removed through a corneal incision above by the small magnet.

24.viii.03. V. = 6/12. Lens clear.

25.x.07. Dr. McKenzie of Torquay has kindly supplied me with the following note. Left eye. Linear scar of cornea down and in just below pupillary margin (site of entry of foreign body). At corneo-scleral margin down and out another linear scar (operation wound no doubt). The eye otherwise normal. V. = 6/6. With + 0·5 D sph. = 6/5 pt. J. 1.

Right eye. Normal. V. = 6/6. With + 0·5 D sph. = 6/5. J. 1.

Case 18.—Robert S., aged 33, under Mr. Fisher. Several days before admission, September 5, 1903, the left eye was injured by a flying chip. The Haab magnet caused the iris to be bulged below. An incision was then made in the sclera below 6 mm. behind the cornea, but the small magnet failed.

16.ix.03. Needling.

19.ix.03. Curette evacuation. The foreign body removed with the small magnet.

17.x.03. Division of anterior synechia.

20.i.04. Vision with glass = 6/9.

Dr. Rockliffe, of Hull, has kindly sent me a note of the present state.

11.xi.07. Left eye. Vision $\bar{c}$ + 12 D sph. = 6/18 badly. Retinal veins full and tortuous. Thin nebula of cornea. Pupil normal. Media clear. Field full.

Right eye. V. = 6/12. Retinal veins full and tortuous.

Case 19.—Thomas E., aged 28, under Mr. Fisher. Three weeks before admission, October 23, 1903, the left eye was injured whilst he was punching. There was a wound of the sclerotic below. V. = 6/18. The X-rays showed the presence of a foreign body in the vitreous.

24.x.03. The Haab magnet failed.

4.xi.03. The foreign body was removed through a scleral puncture by the small magnet.

14.xi.03. Vision = hand movements. Bad reflex. Many vitreous opacities.

19.xi.03. Left. T. −. Almost universal detachment of retina.

Case 20.—William S., aged 34, under Mr. Gunn. A week before admission, October 24, 1903, whilst chipping a chain, a fragment flew into his right eye. There was a wound of the cornea up and in, a hole in the iris, and an opaque lens. The foreign body was drawn forward with the Haab magnet, and became entangled in iris. A peripheral section was then made with a keratome and a piece of steel removed by the small magnet.

Iridectomy was then performed.

27.x.03. Iris adherent to the wound of operation.

19.vi.07. Right eye. Quiet. No injection. Good anterior chamber. Both pillars of coloboma adherent to scar. Pupil dilates well. Lens clear. One large vitreous opacity far back. Fundus normal.

V. = 6/12. $\bar{c}$ $\left.\begin{matrix} -1 \text{ D sph.} \\ -1 \text{ D cyl. Ax. } 75^\circ \text{ down and in} \end{matrix}\right\}$ 6/6 pt. J. 1.

Left eye. Normal. V. = 6/5. J. 1.

Case 21.—Stephen U., aged 36, under Mr. Collins. Five days previous to admission, November 21, 1903, the right eye was injured by a flying fragment from a neighbouring bench at his work. There was a wound at the corneo-scleral margin

down and in, iritis, the lens clear, and much vitreous opacity. The foreign body was localised by the X-rays in the bottom of the vitreous.

23.xi.03. The foreign body was removed through a scleral incision below by the small magnet.

21.xii.03. Right. Vision = hand movements. Projection bad.

27.viii.07. Right eye. Has given no trouble. Quiet. No K.P. Iris bright, dilates well. Some streaky opacity in the posterior part of the lens. Large vitreous opacities. Far forwards just behind the site of entry of the foreign body and magnet a large white mass can be seen by focal illumination. Behind this a fairly extensive grey detachment of the retina drawn forward to the above-mentioned mass. There are several small white patches of choroidal atrophy scattered about the retina. T. n. V. = 6/60.

Left eye. Normal. V. = 6/5.

Case 22.—Bertie V., aged 14, under Mr. Tay. Four days before admission, December 21, 1903, the left eye was injured whilst he was chipping. There was a wound of the cornea, up and out, hypopyon and a small wound of the lens up and out. The foreign body was removed by the Haab magnet through the original wound.

3.ii.04. V. = 6/9.

4.ii.04. Division of anterior synechia.

19.vi.07. Left eye. Quiet. No K.P. Small corneal scar up and out. One posterior synechia to wound in lens, the opacity in which has remained quite stationary. No vitreous opacity. Fundus n.

V. = 6/12. $\bar{c}$ $\begin{matrix} -0{\cdot}25 \text{ D sph.} \\ +2 \text{ D cyl Ax. 20 down and out} \end{matrix}$ } 6/5 ptly. J. 1.

Right eye. Normal. V. = 6/5 J. 1.

Case 23.—Fred. G., aged 18, under Mr. Fisher. On the day of admission, January 1, 1904, whilst he was hammering, the left eye was injured by a flying chip. There was a small corneo-scleral wound to the inner side. A small bead of vitreous was presenting.

20.i.04. Left vision with glass = 6/6 pt.

27.viii.07. Patient does not remember which eye was injured.

Left eye. No injection. There is a small white scar at the corneo-scleral junction on the inner side. No K.P. Pupil dilates well. No synechiæ. Lens clear and in situ. No vitreous opacity. Fundus normal. T. n. V. = 6/6 and J. 1. Right eye normal. V. = 6/5 pt. J. 1.

Case 24.—Arthur F., aged 31, under Mr. Lang. Five weeks before admission, January 21, 1904, the right eye was injured by a chip whilst he was chipping. There was a small scar of the cornea, the iris was fully dilated, and there were fine opacities in the lens in the posterior part up and out.

The foreign body was drawn forwards with the Haab magnet and removed through a corneal incision above, by the magnetised spud.

26.i.04. V. = 6/30. $\bar{c}$ +1 D sph. = 6/18.

16.x.05. Lens opaque. V. = Hand movements. Projection good.

Left eye. V. = 6/6. J. 1.

Case 25.—Forbes M., aged 20, under Mr. Silcock, was admitted May 19, 1904. In November, 1903, the left eye became dim. An X-ray photograph showed a small foreign body in the lens.

There was a small scar in the cornea down and in. The pupil dilated well. The lens was completely opaque. V. = Perception of light. Projection good.

20.v.04. Needling.

21.v.04. Curette evacuation. Small foreign body removed by the small magnet.

10.vi.04. K.P. in the left eye.

8.vii.07. Left eye. Quiet. No injection. Small irregular scar in cornea down and in. Horizontal scar in cornea above (operative). Much fine old K.P. Pupil dilated well. Clear gap in capsular membrane. No vitreous opacity. Fundus normal. V. $\bar{c}$ +11·5 D sph. = 6/5. $\bar{c}$ +15·5 D sph. = J. 1.

Right eye. Normal. V. = 6/5.

Case 26.—Peter S., aged 53, under Mr. Lawford. Eighteen years previous to admission, June 3, 1904, the right eye was

injured by a piece of nail. The eye was quiet until three years ago when it became red and painful. There have been several similar attacks. The eye was quiet, and up and out a small ragged nodule could be seen lying on the iris.

An X-ray examination localised a foreign body in this situation.

4.vi.04. Through a peripheral Graefe section the foreign body was removed by the small magnet.

11.vi.04. Vision = 6/18.

8.vii.07. Right eye. Quiet. Cornea clear. No K.P. Good anterior chamber. Iris bright. A small partial coloboma upwards with the outer pillar adherent to scar. Lens clear. Vitreous clear. Fundus normal. T. n.

V. = 6/60. $\bar{c}$ $\left.\begin{matrix} -3{\cdot}5 \text{ D sph.} \\ \hline -1{\cdot}5 \text{ D cyl. Ax. } 45^\circ \text{ down and in} \end{matrix}\right\}$ 6 6.

$\bar{c}$ $-1{\cdot}5$ D cyl. Ax. 45° down and in. J. 1.

Left eye. Normal. V. = 6/60. $\bar{c}$ $\left.\begin{matrix} -3 \text{ D sph.} \\ \hline -1 \text{ D cyl. Ax. H.} \end{matrix}\right\}$ 6/6.

$\bar{c}$ -1 D cyl. Ax. H. = J. 1.

Case 27.—George W., aged 45, under Mr. Collins. Three years previous to his admission, June 18, 1904, he was struck in the left eye by a flying fragment from a steel tool. The sight failed at the time, but the eye remained quiet up to three weeks ago. There was great injection, some corneal opacity, deep anterior chamber, tremulous iris, and a shrunken opaque lens. A foreign body could be seen at the bottom of the anterior chamber.

20.vi.04. Through a peripheral keratome incision the foreign body was removed by the small magnet. Iridectomy was then performed.

23.viii.07. Left eye. Quiet. Has given no trouble. Much scarring of cornea. No K.P. Good anterior chamber. Iris atrophic. Coloboma down and in, with pillars entangled in scar. Lens shrunken and opaque—much vitreous opacity. Post neuritic optic atrophy. T. n. V. = fingers at 1 metre. Projection good.

Right eye. Several corneal scars V. = 6/12. $\bar{c}$ $+2$ D sph. = J. 1.

Case 28.—Arthur S., aged 24, under Mr. Lang. The day before admission, June 30, 1904, the left eye was injured whilst he was chipping. There was a wound of the upper lid and the sclerotic. A large foreign body was removed through the wound of entry with the small magnet. The conjunctiva was sutured over the wound.

21.vii.04. Many vitreous opacities. A large patch of cicatricial tissue was seen with the ophthalmoscope at the site of injury, V. = 6/18. No scotoma detected with the perimeter.

15.x.07. Left eye has given no trouble, but the sight is not so good. Quiet. A large depressed scar of the sclerotic above just anterior to the equator. Cornea clear. No K.P. Fine cobweb-like post cortical opacity of the lens. Some vitreous opacity. Optic disc normal. Macula healthy.

The whole of the upper half of the retina is detached in billowy sections. Up and out in the situation of injury is a large white scar from which the detachment springs, but in the neighbourhood of the scar the detachment is not so marked. In the nasal part the detached retina reaches the lens and can be well seen with +12 D sph. T. n. V. = 6/60.

Right eye normal. V. = 6/5. J. 1.

Case 29.—George S., aged 32, under Mr. Morton. Three months previous to admission, July 15, 1904, the left eye was injured whilst he was picking the road. There was a central scar of the cornea, the lens was clear, there were vitreous opacities. The foreign body was drawn forward by the Haab magnet, and removed through a peripheral incision with the Haab magnet.

29.vii.04. Lens clear. Many vitreous opacities. Vision = 6/9.

20.viii.07. Left eye. Quiet. Cornea clear, but for scar of injury. No K.P. Pupil dilates well, no pigment on lens capsule. Lens clear. Much stringy, coarse, vitreous opacity, most marked in fore part of vitreous. Fundus normal. T. n. V. = 6/9 pt.

Right eye. Normal. V. = 6/6. J. 1.

Case 30.—William C., aged 50, under Mr. Spicer. Five days before admission, August 10, 1904, the left eye was injured by a flying chip. There was intense injection, an abscess of the

cornea and hypopyon. A foreign body was localised by the X-rays in the lower part of the anterior chamber.

17.viii.04. The foreign body was removed by the Haab magnet through the wound of entry.

27.viii.04. V. with glass = 6/12.

23.x.07. Left eye. Quiet. Scar of cornea down and out. No K.P. No synechiæ. Lens clear. No vitreous opacity. Fundus normal. T. n. V. = 6/9. $\bar{c}$ +1 D sph. = 6/6; $\bar{c}$ +3 D sph. = J. 1.

Right eye. Normal. V. = 6/9. $\bar{c}$ +1 D sph. = 6/6; $\bar{c}$ +3 D sph. = J. 1.

Case 31.—Thomas B., aged 43, under Mr. Fisher. On the morning of admission, August 20, 1904, the left eye was injured by a flying fragment. There was a small wound at the corneo-scleral margin on the inner side. Blood in the vitreous. A foreign body was removed by the Haab magnet, a bead of vitreous was cut off. The iris was left entangled in the wound.

9.ix.04. Iridectomy was performed.

27.ix.04. Vision = fingers at 0·5 metre.

27.viii.07. Left eye. Quiet. No injection. Cornea bright. No K.P. Good anterior chamber. Extensive and irregular coloboma inwards. Pillars free. Localised opacity in the lens at site of injury. Much uveal pigment about anterior lens capsule. No vitreous opacity. Fundus not well seen, but no detachment or gross lesion detected. T. n.

V. $\bar{c}$ $\left.\begin{array}{l}+0{\cdot}75 \text{ D sph.} \\ \hline +5 \text{ D cyl. Ax. } 70^\circ \text{ down and in}\end{array}\right\}$ 2/60. Projection fair.

Right eye. Central corneal nebula. V. = 6/18. $\bar{c}$ +1 D sph. = 6/12; $\bar{c}$ +2·5 D sph. = J. 4.

Case 32.—Harry D., under Mr. Fisher. On the morning of admission, September 27, 1904, his right eye was injured by a piece of wire. There was a wound of the cornea down and in, and a foreign body could be seen in the lens. The eye was X-rayed.

28.ix.04. A piece of wire, $\frac{3}{4}$ inch long, was removed by the Haab magnet.

22.x.04. Division of anterior synechia.

27.viii.07. Right eye. Quiet. Has given no trouble Cornea clear, but for a pigmented scar down and in, to which the

iris was adherent. No K.P. Good anterior chamber. Iris bright. One posterior synechia below. Dense opaque capsular membrane. T. n. V. = hand movements. Projection good.

Left eye. Normal. V. = 6/5.

Case 33.—John F., aged 32, under Mr. Lawford. On the day of admission, September 28, 1904, the right eye had been struck by a chip of steel. There was a wound of the sclera up and out, just behind the ciliary region. The lens was clear. There were a few vitreous opacities. No foreign body or detachment seen. The foreign body was localised by the X-rays just behind the lens. A large foreign body was removed through a scleral wound by the small magnet.

13.x.04. Vision = 6/36.

8.vii.07. Right eye. Quiet. Up and out a scar of the conjunctiva, closely bound down to the sclerotic. Cornea clear. No K.P. Good anterior chamber. Pupil active and regular. Some pigment on anterior lens capsule above. Lens clear, *in situ*. One large vitreous opacity, bright and glistening, in mid-vitreous. No detachment seen, but it is not possible to see the fundus so far forward as the scar in the sclera. T. n. V. = 6/5 pt.

Left eye. Normal. V. = 6/5 pt.

Case 34.—William J., aged 22, under Mr. Lawford. On the day of admission, January 10, 1905, the right eye was injured by a flying chip. There was a central wound of the cornea and traumatic cataract. The X-rays localised a minute chip far back in the vitreous.

11.i.05. The Haab magnet produced no effect. The small magnet, introduced through a corneal incision above, ineffectual. Curette evacuation.

14.i.05. The wound was reopened, and the terminal of the small magnet introduced into the vitreous. A minute chip of steel was removed.

23.i.05. Vision with glass = 6/24. No vitreous opacity.

30.vii.07. Right eye. Deviates outwards. Quiet. Central pigmented scar of cornea, and a linear scar above (operative), to which a tag of capsule is adherent. No K.P. Shallow anterior chamber. Pupil active and regular. Fairly opaque

capsular membrane. No vitreous opacity. Fundus normal. No detachment. T. n.

V. $\bar{c}$ $\left.\begin{matrix}+12 \text{ D sph.} \\ \hline +1{\cdot}5 \text{ D cyl. Ax. } 30^\circ \text{ down and in}\end{matrix}\right\}$ 6/24. Adde + 4 D sph. = J. 2.

Left eye normal but for a small patch of pigment on anterior lens capsule. V. = 6/9.

Case 35.—Ernest G., aged 22, under Mr. Lang. On the day of admission, February 13, 1905, the left eye was injured whilst he was chipping. There was a central wound of the cornea, and a foreign body could be seen in the lens. A corneal incision was made below, and a piece of steel, 7 mm. × 2 mm. × 0·5 mm., was removed by the magnetic spud.

20.ii.05. Curette evacuation.

11.v.05. Needling.

20.v.05. V. $\bar{c}$ $\left.\begin{matrix}+10 \text{ D sph.} \\ +1 \text{ D cyl. Ax. H.}\end{matrix}\right\}$ 6/18. Adde 4 + D sph. = J. 1.

8.vii.07. Left eye. Deviates outwards. Central corneal scar; also two horizontal scars, one above and the other below the centre of the cornea. No K.P. Iris tremulous. Clear gap in capsular membrane. No vitreous opacity. Fundus normal. T. n.

V. $\bar{c}$ $\left.\begin{matrix}+9 \text{ D sph.} \\ +2 \text{ D cyl. Ax. H.}\end{matrix}\right\}$ 6/12. Adde + 4 D sph. = J. 4.

Right eye. Normal. V. = 6/5. J. 1.

Case 36.—Charles M., aged 17, under Mr. Lang. On the day of admission, May 10, 1905, the left eye was injured whilst he was assisting at rivetting. There was a corneal wound down and in and a piece of metal could be seen running straight backward and pinning back the iris. Lens clear.

11.v.05. A needle-shaped piece of steel was removed by the Haab magnet.

18.v.05. Vision = 6/9. $\bar{c}$ + 1 D sph. = 6/5.

14.x.07. Right eye. Normal. V. = 6/6. J. 1. Left eye. Normal. V. = 6/6. J. 1.

Case 37.—Richard B., under Mr. Collins. Six days before admission, May 15, 1905, the right eye was injured by a flying chip.

There was a wound of the corneo-scleral margin to the inner side, vitreous was presenting, the lens was clear, no vitreous opacity. The X-rays localised a foreign body in the vitreous. The Haab magnet produced great pain.

18.v.05. Through a scleral puncture in the situation of localisation, the foreign body was removed with the small magnet.

25.v.05. Vision = 6/60. Inner part of retina detached.

15.vii.05. Eye quiet. T. low n.

Case 38. James V., aged 35, under Mr. Lang. The day of admission, May 29, 1905, whilst he was chipping, the left eye was injured. The right eye had been excised for a similar accident in 1892. In the left there was a central wound of the cornea, the pupil was dilated with atropine, and the lens was injured. A foreign body was localised in the vitreous by the X-rays, and the chip removed by the Haab magnet the same night.

5.vi.05. Extraction with iridectomy.

19.vi.05. Division of anterior synechia.

5.vii.05. Vision = bare perception of light. Projection bad. A yellow mass seen to occupy the vitreous.

27.x.05. As before. V. = hand movements. Projection bad.

Case 39.—Ellen W., aged 49, under Mr. Spicer. Five days before admission, July 6, 1905, whilst she was hammering an iron bedstead the left eye was injured. There was a wound of the cornea, and traumatic cataract. A foreign body was localised by the X-rays far back in the vitreous.

7.vii.05. The Haab magnet withdrew the foreign body.

18.vii.05. Curette evacuation.

20.vii.05. Division of anterior synechia.

3.vii.05. Vision = bare perception of light. Projection bad.

14.x.07. Left eye. Quiet. Central corneal scar. No K.P. Extensive posterior synechiæ. Dense pigmented capsular membrane. T. n. V. = hand movements. Projection fair.

Case 40.—William B., aged 19, under Mr. Morton. Two and a-half months before admission, October 21, 1905, he had been struck in the left eye whilst paring a horse's hoof with a hammer and knife. There was a small scar in the outer part

of the cornea, partial traumatic cataract, and many vitreous opacities. There was slight optic neuritis, and down and out a mass of exudate in the retina, in which a foreign body could be seen. V. = 6/36. The X-rays confirmed this.

24.x.05. The Haab magnet was applied unsuccessfully.

20.viii.07. Left eye has remained quiet since his discharge. Quiet. No injection. Cornea clear, but for a small pigmented scar to which a knuckle of iris is adherent. No K.P. Pupil dilates fully. Lens clear. No vitreous opacity. Running from the wound in the cornea directly backwards is a silvery rod of fibrous tissue. This becomes connected with a cone of detached retina, over which vessels can be seen coursing. There is a solitary star-shaped mass of pigment to the outer side of this. Nothing is seen suggesting a foreign body.

The optic disc shows a considerable mass of connective tissue down and in, otherwise it is a good colour. Macula perfect. V. = 6/5 and J. 1.

Right. Eye normal. V. = 6/5 J. 1.

Case 41.—Teddy W., aged 18, under Mr. Lang. Seven days before admission, November 9, 1905, whilst cutting a rivet with a hammer and chisel the right eye was injured. There was a wound of the cornea, and traumatic cataract. T. +1. A foreign body was localised in the lens by the X-rays.

13.xi.05. The foreign body was drawn forward by the Haab magnet and removed through a corneal incision above by the magnetised spud. Curette evacuation.

14.xi.05. Division of anterior synechia.

27.vii.07. Right eye. Quiet. Cornea clear, but for a scar just to the outer side of the centre, and the operation wound above. No K.P. Iris tremulous. No synechiæ. Good gap in opaque capsular membrane. No vitreous opacity. Fundus normal. T. n.

V. $\bar{c}$ $\left.\begin{matrix} +9 \text{ D sph.} \\ +4 \text{ D cyl. Ax. V.} \end{matrix}\right\}$ 6/6. Adde + 4 D sph. = J. 1.

Left eye. Normal. V. = 6/6 J. 1.

Case 42.—William P., aged 25, under Mr. Lang. The day before admission, November 25, 1905, the right eye was injured whilst he was using a hand drill. There was a corneal wound down

and in, the cornea was hazy. A wound of the iris and lens. V. = 6/9. The foreign body was localised in the lens by the X-rays.

27.xi.05. The foreign body was drawn forward with the Haab magnet and removed by the small magnet through a corneal incision above.

19.xi.05. Quiet, well dilated, the opacity of the lens has increased. V. = Fingers at 2 metres.

5.xi.06. Right eye. Quiet. No K.P. Complete cataract. V. = Fingers at 25 cm. Projection excellent. T. n.

Up to the present considerable stress has been laid upon the relative merits of the Haab or small magnet in the extraction of foreign bodies from within the globe. Both instruments are indispensable, and there are cases suitable for each. For instance, suppose a foreign body be situated far back in the lens, no one would thrust the terminal of the small magnet into the eye without first releasing the foreign body and bringing it into the anterior chamber by the Haab magnet, so that it could then be gently removed with the small magnet. Thus a consideration, as it were, of Haab magnet *versus* small magnet is beside the point. What is really of importance is the point of entry the foreign body took and the path it pursued within the globe, and whether the foreign body was removed through the cornea, or by way of a scleral puncture. The cases, therefore, will be treated with these considerations as a basis, and they will be arranged as follows :—

A. Cases in which the foreign body was removed by way of the cornea.

B. Cases in which the foreign body was removed by scleral puncture.

In the sub-sections notice will be taken as to whether the lens was injured, and the final situation of the foreign body.

As regards the ultimate vision obtained, the divisions have been necessarily rough to prevent unnecessarily

splitting up the cases, and so spoiling any broad conclusions that may be drawn. Therefore, the following plan has been adopted. Cases giving:—

(1) Vision of at least 6/12, and not capable of further improvement.

(2) Vision of less than 6/12, and capable of further improvement by operation as, for example, the needling of an opaque membrane or the extraction of an opaque lens.

(3) Vision less than 6/12, and not capable of improvement. This group includes the cases in which vision has been almost or quite lost, but in which excision was not thought necessary.

A. Cases in which the Foreign Body was Removed Through the Cornea or Corneo-Scleral Junction.

Path of Entry Through the Cornea or Corneo-Scleral Margin.

1. Foreign body situated anterior to the vitreous with the lens uninjured. Five Cases. Nos. 7, 14, 26, 30, 36.

Results—

(*a*) Not less than 6/12, and not capable of improvement by further operation. Five cases = 100 per cent.

(*b*) No cases.

(*c*) No cases.

2. Foreign body situated anterior to the vitreous with the lens injured. Twelve Cases. Nos. 4, 5, 6, 13, 15, 22, 24, 25, 27, 35, 41, 42.

Results—

(*a*) Not less than 6/12, and not capable of improvement by further operation. Six cases = 50 per cent.

(*b*) Less than 6/12, and capable of improvement by further operation. Three cases = 25 per cent.

(*c*) Less than 6/12, and not capable of improvement by further operation. Three cases = 25 per cent.

3. Foreign body situated in the vitreous with the lens uninjured. Six cases. Nos. 11, 17, 20, 23, 29, 40.

Results—

(*a*) Not less than 6/12, and not capable of improvement by further operation. Five cases = 83·4 per cent.

(*b*) No cases.

(*c*) Less than 6/12, and not capable of improvement by further operation. One case = 16·6 per cent.

4. Foreign body situated in the vitreous with the lens injured. Ten cases. Nos. 1, 3, 9, 12, 16, 31, 32, 34, 38, 39.

Results—

(*a*) Not less than 6/12, and not capable of improvement by further operation. Five cases = 50 per cent.

(*b*) Less than 6/12, and capable of improvement by further operation. Three cases = 30 per cent.

(*c*) Less than 6/12, and not capable of improvement by further operation. Two cases = 20 per cent.

B. Cases in which the Foreign Body was Removed from the Vitreous by Scleral Puncture.

I. *Path of Entry Through the Cornea or Corneo-Scleral Margin.*

1. Lens uninjured. Three cases.

Results—

(*a*) No cases.

(*b*) No cases.

(*c*) Less than 6/12, and not capable of improvement by further operation. Three cases = 100 per cent.

2. Lens injured. Two cases.

Results—

(*a*) No cases.
(*b*) No cases.
(*c*) Less than 6/12, and not capable of improvement by further operation. Two cases = 100 per cent.

II. *Path of Entry by Sclerotic, and Removed either Through Wound of Entry or Scleral Puncture.*

Four cases.

Results—

(*a*) Not less than 6/12, and not capable of improvement by further operation. One case = 25 per cent.
(*b*) No cases.
(*c*) Less than 6/12, and not capable of improvement by further operation. Three cases = 75 per cent.

Conclusions.

Although these cases support what in many instances we should anticipate, a few very striking features are apparent. In those cases in which the foreign body entered the eye by way of the cornea, almost everything depends upon whether the lens was intact or injured.

In no case in which the foreign body was anterior to the vitreous and the lens uninjured was it found necessary to excise the eye, and in the cases of this series none gained less than 6/12 and many full vision. Again, those cases in which the foreign body was situated in the vitreous (having gained that situation by the same path) and the lens intact give a better result than those in which the foreign body was embedded in the lens.

The cases that give the ultimate worst result are those in which the foreign body entered the globe by the sclerotic,

or in which a piece of steel was removed by scleral puncture. In the solitary case giving 6/5 of vision, the foreign body entered just outside the corneo-scleral margin, and presumably pierced the retina anterior to the ora serrata, where a wound would not be so serious as one nearer the equator.

In the appendix will be found tabulated the cases which I have not been able to trace, and those in which it was found necessary to excise the globe. Although it was not intended to include any cases in which a final result was not known, or which had not been examined at least a year after the injury, yet it seemed a pity not to place the material collected on record. The list of cases of excision at least teaches something, and supports the fact that damage to the lens is an extremely serious complication in injuries to the globe by foreign bodies. Where known, the reason for excision has been stated.

Finally, I have to express my thanks to the surgeons of the hospital, who have been so kind as to allow me to use their cases as material for this paper.

TABLE OF CASES DIS-

No.	Name.	Surgeon.	Sex.	Age.	Eye.	Date of injury.	X-rays.	Site.	Magnet used.
1	Wm. D. ...	Mr. Lang ...	M.	18	L.	3 days	Yes	Vitreous	Small
2	Wm. S. ...	,, Collins ...	M.	38	L.	15 months	Yes	,,	Haab and Small
3	Wm. W. ...	,, Spicer ...	M.	17	R.	7 ,,	Yes	,,	,, ,,
4	Robt. H. ...	,, Fisher ...	M.	25	L.	1 day	No	Lens	Haab
5	Hy. B. ...	,, ,, ...	M.	18	L.	9 weeks	Yes	,,	Haab and Small
6	H. B.	,, Silcock ...	M.	36	L.	12 hours	No	A.C.	Small
7	Jas. P. ...	,, Collins ...	M.	37	L.	1 day	No	,,	,,
8	Wm. S. ...	,, Lawford	M.	?	R.	5 weeks	Yes	Vitreous	Haab and Small
9	Hy. S. ...	,, Spicer ...	M.	18	R.	5 ,,	Yes	,,	,, ,,
10	Wm. L. ...	,, Collins ...	M.	15	L.	1 year	No	,,	Small
11	Hy. R. ...	,, Spicer ...	M.	45	L.	Same	No	,,	Haab
12	Alb. D. ...	,, Morton ...	M.	35	L.	8 days	Yes	,,	Haab and Small
13	Chas. M. ..	,, Lawford	M.	22	L.	1 month	No	Lens	Small
14	Jno. P. ...	,, Collins ...	M.	31	L.	6 days	No	A.C.	Haab and Small
15	Herb. C. ...	,, Silcock ...	M.	?	L.	Same	No	Lens	,, ,,
16	Chas. B. ...	,, Tay ..	M.	22	R.	Same	No	Vitreous	,, ,,
17	Geo. T. ...	,, Collins ...	M.	38	L.	1 day	Yes	,,	,, ,,
18	Robt. S. ...	,, Fisher ...	M.	33	L.	days	No	,,	,, ,,
19	Thos. E. ...	,, ,, ...	M.	28	L.	3 weeks	Yes	,,	Small
20	Wm. S. ...	,, Gunn ..	M.	34	R.	3 days	No	,,	Haab and Small
21	Stephen U. ...	,, Collins ...	M.	36	R.	5 ,,	Yes	,,	Small
22	Bertie V. ...	,, Tay ...	M.	14	L.	8 ,,	Yes	Lens	Haab
23	Fred. G. ...	,, Fisher ...	M.	18	L.	Same	Yes	Vitreous	,,
24	Arthur F. ...	,, Lang ...	M.	31	R.	5 weeks	No	Lens	Haab and Small
25	Forbes M. ...	,, Silcock ...	M.	20	L.	?	Yes	,,	Small
26	Peter S. ...	,, Lawford	M.	53	R.	18 years	Yes	A.C.	,,
27	Geo. W. ...	,, Collins ...	M.	45	L.	3 ,,	No	,,	,,
28	Arthur S. ...	,, Lang ...	M.	24	L.	1 day	No	Vitreous	,,
29	Geo. S. ...	,, Morton ...	M.	32	L.	3 months	No	,,	Haab and Small
30	Wm. C. ...	,, Spicer ...	M.	50	L.	5 days	Yes	A.C.	Haab
31	Thos. B. ...	,, Fisher ...	M.	43	L.	Same	No	Vitreous	,,
32	Harry D. ...	,, ,, ...	M.	?	R.	Same	Yes	,,	,,
33	Jno. F. ...	,, Lawford	M.	32	R.	Same	Yes	,,	Small
34	Wm. J. ...	,, ,,	M.	22	R.	Same	Yes	,,	,,
35	Ernest G. ...	,, Lang ...	M.	22	L.	Same	No	Lens	,,
36	Chas. M. ...	,, ,, ...	M.	17	L.	Same	No	A.C.	Haab
37	Rich. B. ...	,, Collins ...	M.	?	R.	6 days	Yes	Vitreous	Small
38	Jas. V. ...	,, Lang ...	M.	35	L.	Same	Yes	,,	Haab
39	Ellen W. ...	,, Spicer ...	F.	49	L.	5 days	Yes	,,	,,
40	Wm. B. ...	,, Morton ...	M.	19	L.	2½ months	Yes	,,	,,
41	Edw. W. ...	,, Lang ...	M.	18	R.	7 days	Yes	Lens	Haab and Small
42	Wm. P. ...	,, ,, ...	M.	25	R.	1 day	Yes	,,	,, ,,

SED IN THIS PAPER.

n of foreign body.	Method of removal.	Further operation.	Final vision.	Notes.
ea, iris, and lens...	Through wound ...	Curette evacuation, paracentesis, needling, iridotomy.	6/9	F.B. still in globe.
ea	Scleral incision ...	... ...	No P.L.	Unsuccessful.
ea and lens ...	Through cornea ...	Curette evacuation ...	6/6	
,, ...	,, ,, ...	Iridectomy, anterior synechia, needling	6/24	
,, ...	,, ,, ...	Scoop extraction	6/18	
ea	Peripheral section	... ...	6/9	Localised lens opacity. Haab failed.
... ...	Through wound ...	... ...	6/5	
ea and lens ...	Scleral puncture ...	... ...	6/18	Vitreous hæmorrhages. Haab failed.
eo-scleral and lens	Through cornea ...	Needling	6/12	Vitreous opacity.
otic	Scleral puncture ...	... ...	No P.L.	Seen in retina.
eo-scleral	Through wound ...	Iridectomy	6/36	Fibrous tissue in vitreous.
ea and lens ...	Through cornea ...	Needling (2), curette ...	6/12	
,, ...	,, ,, ...	Curette evacuation ..	6/36	Corneal scar.
ea	Peripheral section	... ...	6/6	
ea, iris, and lens...	Through wound ...	Iridectomy	Fingers. Proj. good	Opaque lens.
,, ,, ...	,, ,, ...	Needling, curette evacuation.	6/5	
ea	Through cornea ...	... ...	6/5	
ea and lens ...	Scleral puncture ...	Needling, curette, anterior synechia.	6/18 pt.	
otic	,, ,, ...	... ...	...	Detachment.
ea and iris... ...	Peripheral section	... ...	6/6	Vitreous opacity.
eo-scleral	Scleral puncture ...	... ...	6/60	Detachment.
ea and lens ...	Through wound ...	Anterior synechia	6/5	Localised lens opacity.
eo-scleral	,, ,,	... ...	6/6 pt.	
ea and lens ...	Through cornea ...	... ...	H.M. Proj. good	Opaque lens.
,, ...	,, ,, ...	Curette, needling	6/5	
ea	Peripheral section	Iridectomy	6/6	
... ...	,, ,, ...	,, ...	No P.L.	Post neuritic atrophy.
otic	Through wound ...	... ...	6/60	Detachment.
ea	Peripheral section	... ...	6/9 pt.	Vitreous opacity.
... ...	Through wound ..	... ...	6/6	
ea, iris, and lens...	,, ,, ...	Iridectomy	2/60	Lens opacity.
ea and lens ...	,, ,, ...	Anterior synechia	H.M. Proj. good	Opaque membrane.
otic	Through wound ...	... ...	6/5	No detachment.
ea and lens ...	Through cornea ...	Curette evacuation ...	6/24	Haab failed.
,, ...	,, ,, ...	Curette, needling	6/12	F.B. 7×2×0·5 mm.
ea	Through wound ...	... ...	6/6	
eo-scleral	Scleral puncture ...	... ...	6/60	Detachment.
ea and lens ...	Through wound ...	Extraction, anterior synechia.	P.L.	Plastic cyclitis.
,, ...	,, ,, ...	Curette, anterior synechia	H.M.	Dense capsule.
ea and iris.. ...	... ...	... ...	6/5	Unsuccessful.
ea and lens ...	Through cornea ...	Curette, anterior synechia	6/6	
,, ...	,, ,, ...	... ...	Fingers. Proj. good	Opaque lens.

APPEN

CASES LAST SEEN LESS THAN A

No.	Name.	Surgeon.	Sex.	Age.	Eye.	Date of injury.	X-rays.	Site.	Magnet.
1	Jas. R. ...	Mr. Tay ...	M.	19	L.	10 days	Yes	Vitreous	Small
2	Alf. H. ...	,, Gunn ...	M.	24	L.	Same	No	,,	,,
3	Sidney H. ...	,, Silcock ...	M.	23	R.	6 weeks	No	A.C.	Haab and Small
4	Edward M. ...	,, ,, ...	M.	19	L.	Same	No	Vitreous	Haab
5	Fred. G. ...	,, ,, ...	M.	?	R.	1 week	Yes	,,	Haab and Small
6	A. L.	,, ,, ...	M.	16	R.	Same	No	Lens	Small
7	Hy. S. ...	,, ,, ...	M.	25	R.	14 days	Yes	,,	Haab
8	Thos. H. ...	,, Collins ...	M.	40	R.	1 day	No	A.C.	,,
9	Thos. A. ...	,, ,, ...	M.	39	L.	Same	Yes	Vitreous	Haab and Small
10	Jesse C. ..	,, ,, ...	M.	22	L.	12 weeks	Yes	Lens	Small
11	Wm. M. ...	,, ,, ...	M.	44	R.	1 day	No	,,	Haab
12	Bernard Y. ...	,, ,, ...	M.	13	R.	7 weeks	Yes	Vitreous	Small
13	Arthur M. ...	,, ,, ...	M.	28	R.	Same	Yes	,,	,,
14	Jno. C. ...	,, ,, ...	M.	27	R.	3 days	No	A.C.	,,
15	Harry W. ...	,, ,, ...	M.	36	R.	?	Yes	Lens	,,
16	Thos. G. ...	,, Fisher ...	M.	71	R.	4 months	Yes	Vitreous	Haab
17	Walter D. ...	,, Flemming	M.	21	L.	Same	Yes	,,	,,
18	Hy. F. ...	,, ,,	M.	32	R.	Same	No	,,	,,
19	Sidney H. ...	,, ,,	M.	20	L.	Same	No	,,	,,
20	Alf. P. ...	,, Lawson ...	M.	24	R.	1 day	No	A.C.	,,
21	James L. ...	,, ,, ...	M.	27	R.	1 day	No	,,	,,
22	James T. ...	,, Lawford ...	M.	27	L.	18 months	Yes	Vitreous	Small
23	Hy. T. ...	,, Spicer ...	M.	17	R.	3 months	No	,,	Haab and Small
24	Jno. B. ...	,, ,, ...	M.	32	R.	2 days	No	A.C.	Haab
25	Jno. M. ...	,, ,, ...	M.	43	L.	1 day	No	Vitreous	,,
26	H. P.	,, Morton ...	M.	23	L.	5 days	Yes	,,	Haab and Small
27	Jno. H. ...	,, ,, ...	M.	36	L.	6 days	No	Lens	,, ,,
28	Thos. McE. ...	,, ,, ...	M.	17	L.	1 day	Yes	Vitreous	Small
29	Alf. D. ...	,, ,, ...	M.	29	L.	2 weeks	No	Lens	Haab and Small
30	Jas. K. ...	,, ,, ...	M.	57	R.	3 days	No	Vitreous	Haab
31	Arthur S. ...	,, ,, ...	M.	18	L.	3 weeks	No	Lens	Haab and Small
32	Geo. B. ...	,, ,, ...	M.	38	R.	?	No	?	,, ,,
33	Wm. S. ...	,, ,, ...	M.	19	R.	6 months	Yes	Lens	Haab
34	Jno. L. ...	,, ,, ...	M.	—	L.	1 day	No	Vitreous	Haab and Small
35	Alf. P. ...	,, Lang ...	M.	28	R.	Recent	No	,,	Small
36	Geo. H. ...	,, ,, ...	M.	22	L.	Same	No	,,	Haab
37	Wm. B. ...	,, ,, ...	M.	39	R.	1 month	No	?	,,
38	Julian M. ...	,, ,, ...	M.	19	R.	1 day	No	Vitreous	Haab and Small

DIX II.

INJURED EYE WAS EXCISED.

Path of foreign body.	Method of extraction.	Further operation.	Reason for excision.
Cornea and lens	Through cornea.		
,, ,, ...	Scleral puncture ...	... ...	Unsuccessful.
,, ,, ...	Through wound.		
,, ,, ...	,, ,, ...	... ...	Panophthalmitis.
,, ,, ...	,, ,, ...	Iridectomy.	
,, ,, ...	,, ,, ...	Anterior synechia.	
,, ,, ...	,, ,, ...	Curette evacuation...	,,
,, ,, ...	,, ,, ...	A.C. washed out	,,
,, ,, ...	Scleral puncture ...	... ...	Unsuccessful.
Corneo-scleral	Through wound ...	Iridectomy	Very large foreign body
Cornea and lens	,, ,, ...	Needling, curette, anterior synechia	Glaucoma.
?	Scleral puncture.		
Cornea and lens	Through cornea ...	Curette	Infection.
,, ,, ...	Through wound.		
,, ,, ...	Scleral puncture ...	... ...	Unsuccessful.
,, ,, ...	Cornea	Needling, anterior synechia ...	No P.L.
,, ,, ...	,, ...	Iridectomy, anterior synechia ...	Irritable.
,, ,, ...	Through wound ...	Curette, parcentesis	,,
,, ,, ...	,, ,, ...	Iridectomy	Glaucoma.
,, ,, ...	,, ,, ...	... ...	Irritable.
,, ,, ...	,, ,, ...	... ...	Hopeless.
,, ,, ...	Peripheral section...	Iridectomy	Unsuccessful.
,, ,, ...	Cornea	Anterior synechia.	
,, ,, ...	Peripheral section...	Curette	Cyclitis.
,, ,, ...	Scleral puncture.		
Sclerotic	,, ,, ...	... ...	Irritable.
Cornea and lens	Through wound ...	Extraction.	
,, ,, ...	Cornea.		
,, ,, ...	Scleral puncture ...	... ...	Unsuccessful.
,, ,, ...	Through wound ...	Iridectomy.	
?	?	Vitreous escaped.	
Cornea and lens	Through wound ...	Iridectomy.	
,, ,, ...	Through cornea ...	,, ...	Panophthalmitis.
,, ,, ...	,, ,, ...	... ...	,,
Corneo-scleral	Scleral puncture ...	... ...	Not successful.
Cornea and lens	Through wound ...	Curette, anterior synechia ...	Irritable.
Cornea, iris, and lens ...	Cornea	... ...	Panophthalmitis.
Cornea and lens	Through wound.		